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Southern Medical Association, New Orleans, Louisiana, November 30 - December 1-2-3, 1937

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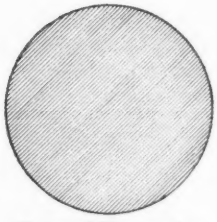

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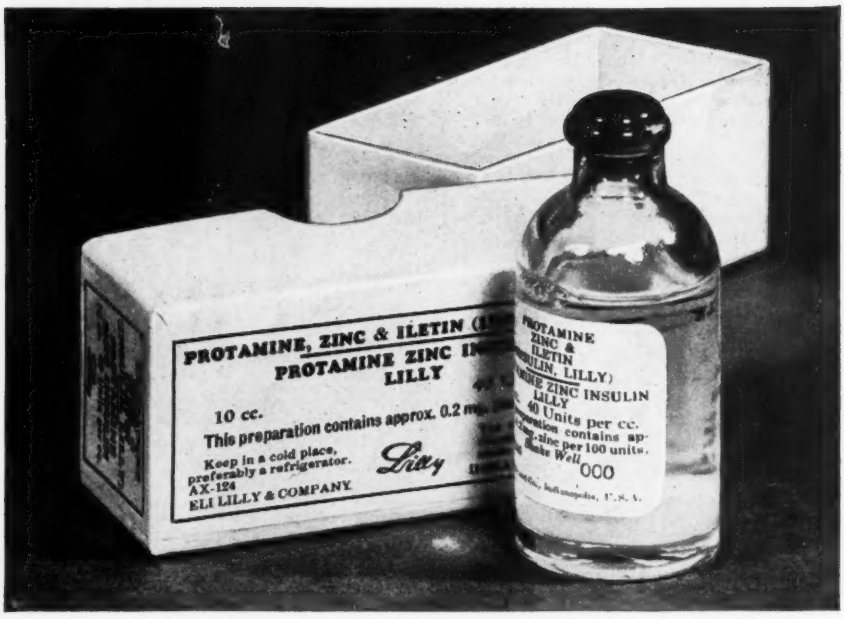

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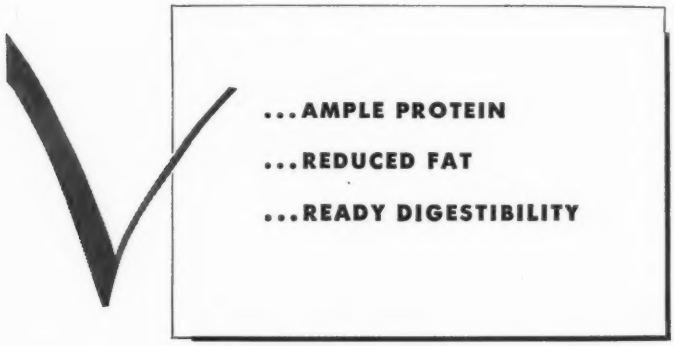

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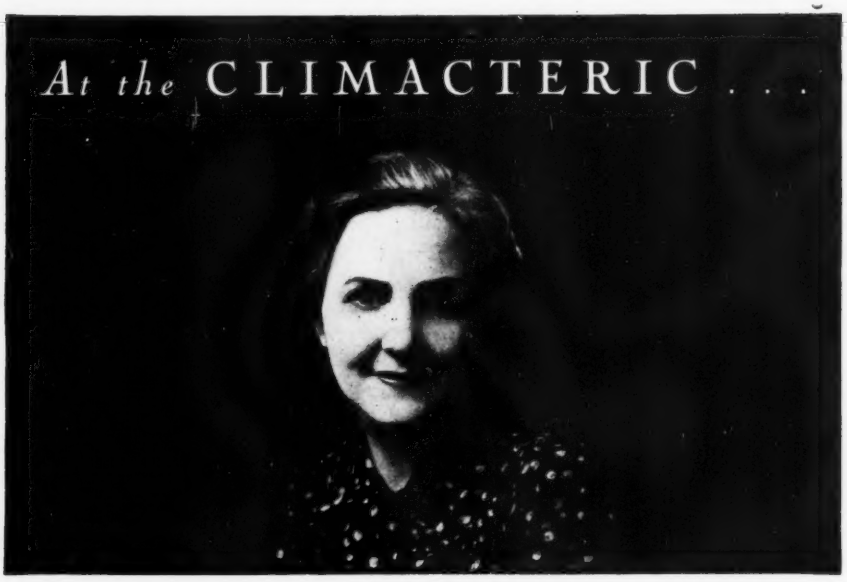

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(1) N. Y. S. Jl. of Med., Oct. 1, 1936 (2) Med. Clin. of N. A., July, 1935

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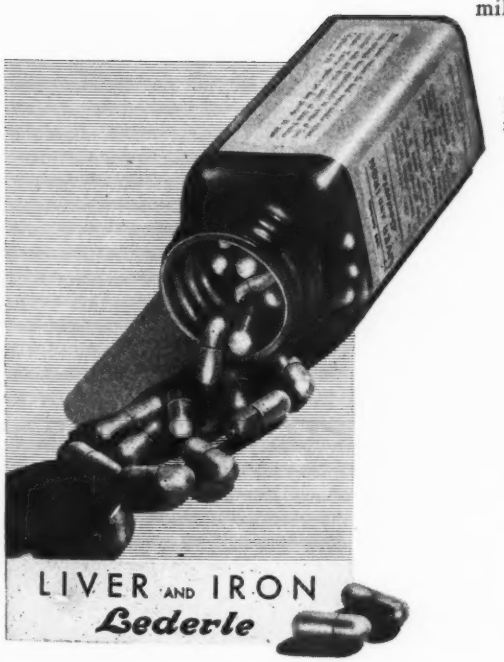

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Water (boiled, then cooled) . . . 16 ounces

Give one to three ounces every hour or two until the stools lessen in number and improve in character.

The mixture may then be strengthened by the gradual substitution of boiled skimmed milk for water until the quantity of skimmed milk is equal to the normal quantity of milk used in the baby's formula. Finally the fat of the milk may be gradually replaced by skimming less and less cream from the milk.

Samples sent to physicians upon request.

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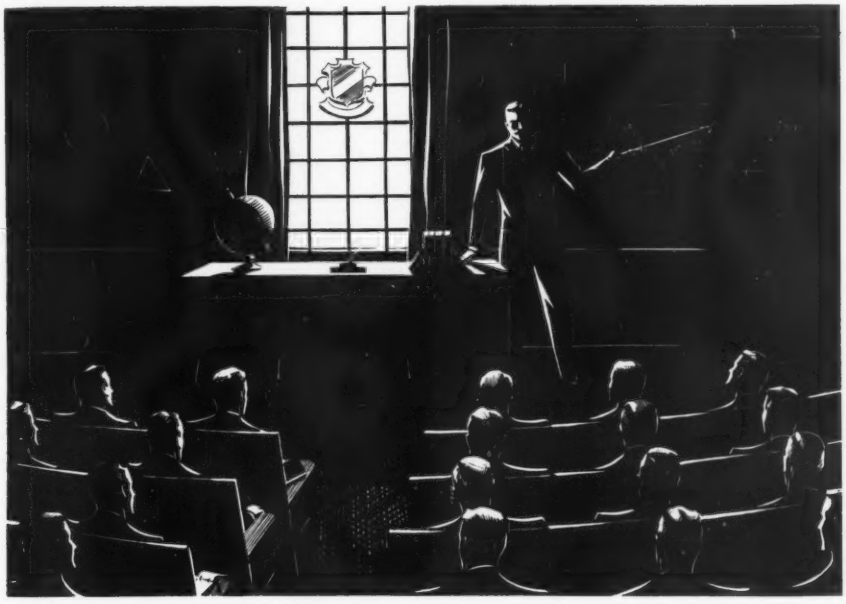

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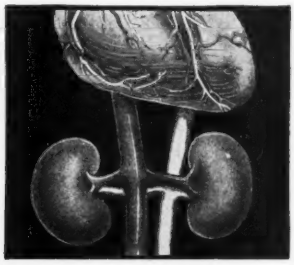

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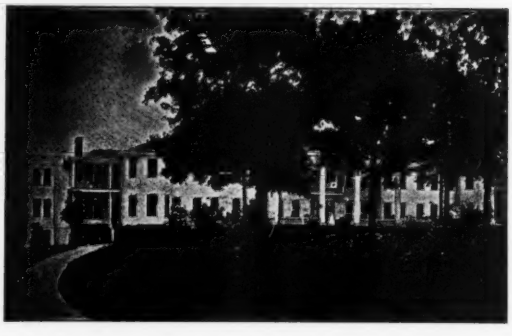

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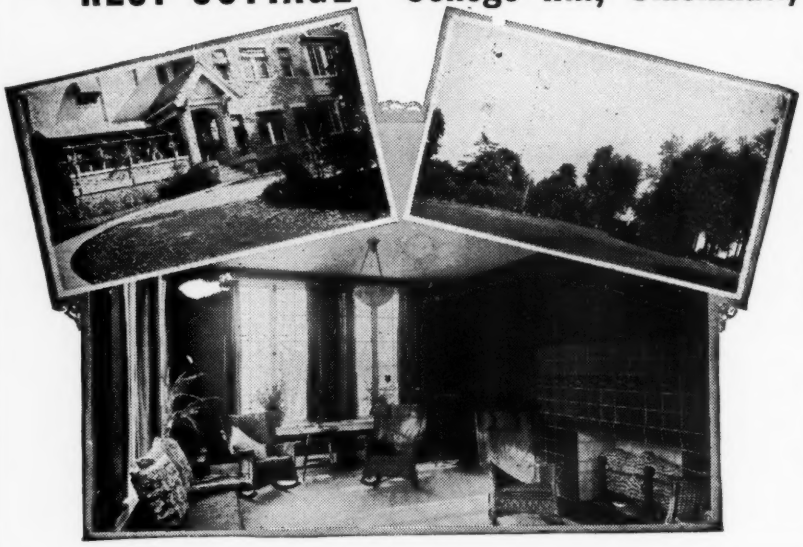

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JOURNAL OF THE SOUTHERN MEDICAL ASSOCIATION

PUBLISHED MONTHLY BY THE SOUTHERN MEDICAL ASSOCIATION AT BIRMINGHAM, ALA.

Volume 30 AUGUST 1937 Number 8

TUBERCULOSIS IN CHILDHOOD*

By J. BUREN SIDBURY, M.D.
Wilmington, North Carolina

Tuberculosis in childhood is a preventable as well as a communicable disease. To combat this disease successfully it must be attacked mainly from this standpoint. It is our prime duty wherever and whenever possible to keep our children from becoming infected.

Certain fundamentals are unquestioned, to wit:

(1) A tuberculous mother should not be allowed to nurse her baby. The baby should never be allowed to go to the breast and the mother should have nothing to do with caring for the baby. Those who take care of the baby must be free of tuberculosis. There is nothing to be gained by taking the baby from a tuberculous mother and allowing the aunt or grandmother who has a chronic cough, possibly tuberculous, to take care of the baby.

(2) When it is possible to make another provision, children should not be allowed to stay in the same house with tuberculous adults. They will sooner or later become infected. When it is impossible to move the children from a tuberculous home, it is imperative that the strictest precautions be observed. The advisability of giving these children B.C.G. vaccination arises, and it should be seriously considered.

(3) Chronic phthisis in the aged is a great menace to infants. Many cases of miliary tuberculosis and tuberculosis meningitis can be traced to a grandparent or aunt, living in the same house, who has a chronic cough. The late Dr. Holt once said that two types of individuals are the commonest sources of danger in tuberculosis: first, the one who thinks he is cured, and second, the one who does not know that he has the disease.

(4) All school teachers should be examined annually. The *Sanatorium Sun,* in the November, 1936, issue says:

"More than eighteen thousand tuberculous teachers are in active service in the schools of America today." In North Carolina, the *Sun* states, "there are possibly 500 teachers now engaged in teaching who have tuberculosis in an active form." The National Tuberculosis Association suggests that "The logical solution will come when all states have laws requiring teachers to present evidence that they are free of tuberculosis in an active form, such evidence to be based upon physical examination supplemented in all cases by x-ray of the chest."

North Carolina has a law requiring a health certificate from every teacher, declaring freedom from tuberculosis or any other communicable disease, to be signed by a reputable physician. The certificate is to be filed with the school official where employed before being allowed to teach. This certificate is to be renewed annually. The law was passed by the 1919 Legislature of North Carolina. How strictly this law is being adhered to can be answered by our school superintendents. I feel that we should have them answer this question annually.

That tuberculosis is a communicable disease of childhood and should be thought of along with such diseases as scarlet fever, diphtheria and whooping cough is proven by the following report of deaths in the State of Michigan:

DEATHS FROM FIVE COMMUNICABLE DISEASES IN MICHIGAN, 1928 TO 1930, INCLUSIVE

	Under 5 Years	Under 15 Years	Under 20 Years
Tuberculosis	522	940	1751
Diphtheria	508	1049	1074
Measles	550	644	657
Whooping cough	623	649	649
Scarlet fever	215	377	376

Tuberculosis should be reported to state health departments as routinely as are the other four

*Read in Section on Pediatrics, Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

diseases mentioned above. Such a procedure will help materially in the reduction of this disease.

Tuberculosis in children usually has an insidious onset, although infants and young children may have a fulminating course. This is frequently determined by the size of the dose of tubercle bacilli and how frequently it is repeated.

The general practitioner is going to see the majority of these early cases and it is desirable that he recognize them early and institute proper treatment, or refer them to a specialist who will give proper supervision.

How can the general practitioner do this?

(1) By doing a careful physical examination on all children brought to him for treatment. Periodic physical examinations are very desirable. The physical examination in the majority of cases will reveal no positive signs.

(2) By doing a tuberculin test.

(3) All patients who show a positive tuberculin test should have an x-ray examination of the chest. At regular intervals, six months to one year, follow-up chest pictures should be made. In case you, as a general practitioner, do not feel competent to read the films and a radiologist is not immediately available, the plan followed by us in North Carolina is very satisfactory. We have x-rays taken and we send them to Dr. P. P. McCain, of the North Carolina Sanatorium. A report of the x-ray findings is sent to the physician. This has been a great help in studying the cases.

These three procedures, which can be done by any general practitioner, will give much assistance in ferreting out early cases of tuberculosis. It will be the means of finding more early cases which might go on to destructive lesions before being diagnosed.

We do not want to wait for the classical signs found in adults, as rales, dullness and hemoptysis. When that stage has been reached little can be done for these patients. If the diagnosis is made early, the majority of the so-called "consumptives" will be prevented.

In the past decade, mesenteric tuberculosis in children has been greatly reduced, due to our careful inspection of dairies. Pasteurization of milk has cut this source of infection to a minimum.

The most common type of infection is from human contacts or open pulmonary tuberculosis in man. The infection is carried into the lungs, down the trachea and bronchial tree to the lung periphery. The tubercle bacilli lodge in the periphery of the lungs near the pleura. According to Chadwick,[4]

"When the tubercle bacillus is implanted in the pulmonary tissue, tubercles form, and about this focus there is a varying amount of inflammation. This perifocal inflammation may be small or it may involve the entire lobe, or a tuberculous pneumonia may result. This latter condition is more often found in the colored race.

"When these tubercles are developing in the lung parenchyma tubercle bacilli are being carried to the regional lymph glands by the lymph stream which drains that area of the lung. Thus we have the parenchymal and tracheobronchial lymph nodes involved in the primary stage. This is the usual occurrence in childhood infection and is sometimes called childhood type tuberculosis or tracheobronchial tuberculosis.

"In the evolution of a tubercle, the center becomes caseous and in this calcium is deposited. Fibrous tissue forms a capsule about the tuberculous nodule as healing takes place. It tends to run a benign course if further infection is prevented. When a child is exposed to further infection, or a spread occurs from its own primary focus, the disease resulting therefrom localizes in the pulmonary parenchyma and does not again involve the tracheobronchial nodes."

Lymph nodes are not always efficient filters. Tubercle bacilli may pass on through the tracheobronchial nodes and reach the thoracic duct, thence to the larger veins and general circulation. When the general circulation is reached, it is quite evident that every organ in the body may become infected by this route. The lymph nodes themselves may become caseous and rupture either into a bronchus or a blood vessel and a massive dissemination and miliary tuberculosis result.

We should consider any tuberculous adenitis as having serious possibilities. If the infection can be confined to the lymph nodes, the patient has a much greater chance of recovery.

Here, I shall define "tuberculous infection" and "tuberculous disease." Tuberculous infection is the state of being infected. The body is harboring the tubercle bacilli, but the body tissues are not altered materially by the presence of the organism.

Tuberculous disease, according to Morse,[5] is "an alteration in the state of the body or of some of its organs, interrupting or disturbing the performance of the vital functions and causing symptoms." In tuberculous disease the presence of the tubercle bacilli has produced pathological changes in the surrounding cells. This may be slight or may progress to the destruction of tissue and cavitation.

INCIDENCE OF TUBERCULOSIS

The incidence of childhood tuberculosis depends upon the exposure to open human cases. It is more common in the congested districts than in rural communities. Poor hygiene is very conducive to its spread. The Negro and Indian races are more susceptible to the disease than whites.

In Europe the statistics from Vienna showed that 50 per cent of the children at 7 years and 95 per cent of those at 12 years gave positive tuberculin reactions. Dr. Morse[5] says: "We are born free of tuberculosis, but in Boston 10 per cent at 1 year and at 16 years 90 per cent are infected, but very few have tuberculous disease. In this country, in the larger cities, like New York, Philadelphia, Boston and San Francisco, the rate is 10 per cent at 2 years, 15 per cent at 4 years, 20 per cent at 7 years, and 40 per cent at 10 years. This rate is much lower than that found in most Southern states."

In North Carolina Dr. P. P. McCain,[14] at the North Carolina Sanatorium, in the past ten years has examined 260,928 children between 6 and 18 years, giving the tuberculin test, using 0.1 milligram of old tuberculin intracutaneously. Fifteen and five-tenths per cent gave positive reactions.

TUBERCULIN STUDY IN NORTH CAROLINA, 1926-1936

Number given tuberculin test	260,928
White	216,849
With 14.6 per cent positive	
Negro	43,620
With 19.7 per cent positive	
Indian	459
With 25.05 per cent positive	
Number of reactors having x-ray	32,920
Number diagnosed by x-ray as having tuberculosis	4,154
Childhood type cases	3,784
Adult type	206
Childhood and adult type	122
Extra pulmonary	33
Extra pulmonary and adult type	6
Classified as suspicious	3,279
Classified as probably negative	25,487

At the Babies' Hospital, Wrightsville Sound, North Carolina, a survey of the records shows that of 312 infants and young children tested, 301 were negative, 6 were two-plus, 4 were three-plus, 1 was four-plus, a percentage of 3.5 positive.

In my office I found the following:

Number of cases given tuberculin test	536
Positive	52
Negative	484
Per cent positive	9

This represents a more selected group of cases, in that they were brought to the office for a careful check-up, in that they had some or all of the usual symptoms of tuberculosis. Of the positive reactors, 18 gave a one-plus reaction; 14 gave a two-plus reaction; 4 gave a three-plus reaction, and 9 gave a four-plus reaction. Of this number only 15 were diagnosed as having tuberculous disease. The ages in this group ranged from 6 months to 16 years.

Since it usually takes 12 to 24 months for calcification of a lymph node to be demonstrated by x-ray,[1] this will emphasize how many cases of early tuberculosis are missed even by x-ray evidence. Every infant or young child who shows a positive tuberculin test should be considered infected and treated accordingly.

TUBERCULIN TEST

The test is done in the same manner as the Schick test. The intracutaneous method of Mantoux is employed. It consists of the injection of 0.1 c. c. of 1-100 or 1-1000 solution of old tuberculin intracutaneously in the inner surface of the forearm. The site of the injection is examined after 48 hours. The reaction is positive if redness and edema are present. If there is no edema, there is no reaction. The

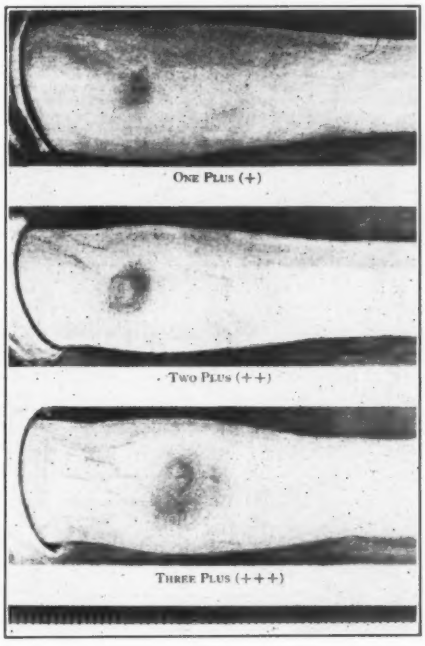

Fig. 1
Tuberculin skin test.

reactions, according to McPhedran,[3] are read one-plus to four-plus. A one-plus reaction is a definite edema, elevated about 1 mm. and not more than 10 mm. in diameter. A two-plus reaction should measure 10 to 15 mm. in diameter and be elevated about 1 mm., with redness beyond the swelling. A three-plus reaction shows edema more than 15 mm., but no necrosis of the skin, and may show some lymphatic involvement radiating from the injection. A four-plus reaction shows extensive edema, redness and a spot of necrosis. Malaise and elevation of temperature may be associated.

The intradermal tuberculin test is much more reliable than the von Pirquet test in that there is a definite amount of tuberculin injected in the skin that must be absorbed and cannot be rubbed off. In a selected group of children 35 per cent gave a positive von Pirquet test, while 53 per cent gave a positive intradermal test.[3] The P.P.D. is the purest product available for giving the test and is freer of false reactions. The cost of this product is its only objection.

The tuberculin test is probably the most important single aid in the early diagnosis of tuberculosis. An infant should give a negative tuberculin reaction. When a positive reaction is shown in an infant or young child, it indicates tuberculous infection of recent date and should always be taken seriously. A positive test does not mean tuberculous disease, nor does it tell where the infection is, or whether active or inactive. It does tell that the patient has living tubercle bacilli present in the body which have grown and liberated tuberculin in the body and that the cells of the body have become sensitive to it. The test should not be limited to infants and children. Its use in adults, if negative, may be of great help in eliminating tuberculosis as a cause of an undiagnosed infection. By means of the tuberculin test, the infected can be separated from the uninfected. This is of tremendous help in ferreting out infected children in a school or family. It is the only reliable method of making an early diagnosis before the appearance of physical signs. Only 12 to 25 per cent of those showing positive tests show evidence in the x-ray. Many cases of acute pulmonary tuberculosis are misdiagnosed and called other pulmonary conditions, even with the aid of the x-ray.

A negative tuberculin test may be obtained in the following conditions:

(1) In uninfected persons.

(2) In overwhelming infection with tuberculosis.

(3) During the incubation period.

(4) In patients treated with tuberculin.

(5) During the acute stage of some contagious diseases, as measles, or scarlet fever.

(6) Unexplained conditions (4 to 8 per cent).

(7) In healed tuberculous lesions.

The interpretation of the tuberculin reaction must depend upon the composite picture of all the available information in the individual case. The x-ray findings, history and physical findings should be considered with the tuberculin reaction. A negative tuberculin test is very strong evidence against tuberculosis.

SYMPTOMS AND PHYSICAL SIGNS

There are no characteristic symptoms of childhood tuberculosis. Dr. Lee Forest Hill[12] says:

"Primary tuberculosis infections are amazingly benign in character. At Lymanhurst a mortality rate of less than 0.5 per cent has been observed in this group of cases. As a rule, the infection occurs, runs its course, and resolves to a calcified focus, fibrotic scar, or disappears altogether, without patient, parent or physician's being aware of what is taking place or has already taken place. In about 75 per cent of primary infections the positive tuberculin test is the only evidence that tubercle bacilli are present somewhere in the body."

The remaining 25 per cent may be demonstrated by x-ray.

The clinical picture of activity may show the following:

(1) Fever. Rectal temperature 100° daily, irregular in type with different level at different times of the day. It is not always high in the afternoons.

(2) Failure to gain in weight at a regular rate. Underweight is not an invariable symptom. It is not unusual to find an overweight child who shows a positive tuberculin skin test.

(3) Languor, easily fatigued, irritability.

(4) These children may be uninterested in life or may be very high strung and very active mentally and physically. Frequently they are very solemn and cannot be made to smile.

(5) Phlyctenules of the cornea.

(6) Tuberculides. These occur on the trunk and exterior surfaces of the extremities.

(7) Delayed recovery from some other illness, as measles, whooping cough, bronchopneumonia or influenza.

(8) Cough and hemoplysis are uncommon symptoms.

Physical signs in childhood tuberculosis are not characteristic and may be found in non-tuberculous conditions equally or more often.

(1) Dullness of chest, rales and increased whispered voice sounds are late signs.

(2) Infected tonsils, carious teeth, intestinal parasites, pyelitis and malaria may cause any of the symptoms associated with tuberculosis.

(3) The tuberculin test and x-ray are the most important signs in the study of tuberculosis.

(4) Sputum examination is not very practical in infants and children. Examination of mucus from the stomach washings before the breakfast meal may be rewarded by the finding of tubercle bacilli.

(5) The x-ray will demonstrate lesions in about 25 per cent of those having positive tuberculin tests.

LOCATE THE CONTACTS

Probably the next most important problem to making a diagnosis of tuberculosis in a child is that of finding the contacts. In fact, the recovery of the infant depends upon how quickly the contact is located and broken. We have many cases of infants who have recovered from tuberculous infiltration of the lung simply because the contact was abruptly stopped before too large and repeated doses of tubercle bacilli were given. It is the duty of every medical man who finds a case of tuberculosis in childhood to study the contacts and protect those exposed to tuberculosis. A search for early cases of tuberculosis should be made by doing tuberculin tests repeatedly on all cases who have been exposed. This is as much our responsibility as giving diphtheria antitoxin to protect against diphtheria. Until we accept this responsibility we shall not meet the situation as we should.

The following cases are presented to demonstrate some of the lesions found in childhood tuberculosis:

Case 2, Fig. 2.—V. S, a white school child 9 years of age, shows multiple primary foci. She has no other symptoms. Her mother died of tuberculosis five years previously. Two sisters are dead of tuberculosis. She is 10 per cent underweight and has no elevation of temperature but shows a three-plus tuberculin reaction.

Case 3, Fig. 3.—M. T., a colored child, 6 years old, who shows a large calcified primary focus in the eighth interspace on the right side and enlarged calcified tracheobronchial lymph nodes in the third and fourth interspaces on the left side. Her father died of tuberculosis two years previously.

Case 4, Fig. 4.—R. G., a colored girl of 8 years, who has childhood type of tuberculosis. Her x-ray shows a calcified primary focus in the third and fourth interspaces and calcified tracheobronchial lymph nodes in the

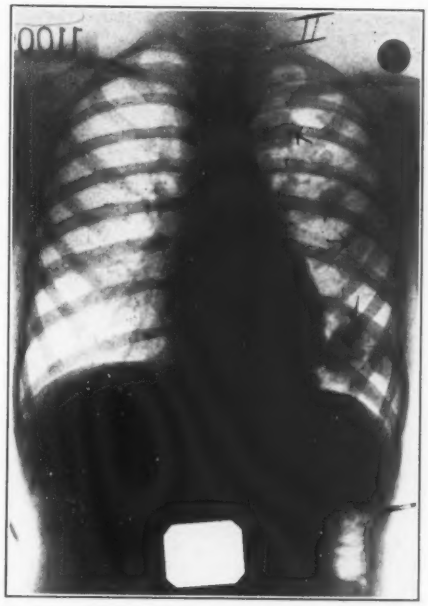

Fig. 2
Primary foci.

Fig. 3
Childhood type tuberculosis.

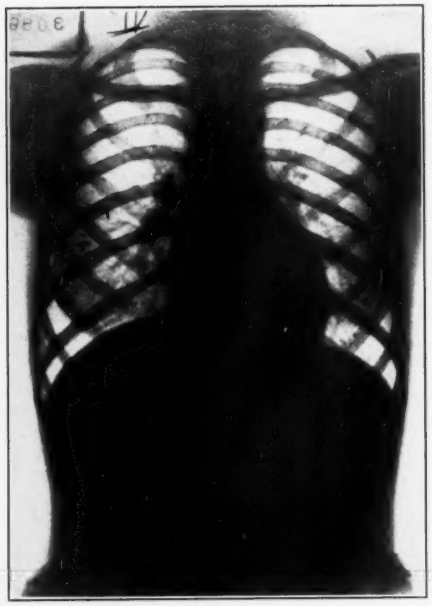

Fig. 4
Childhood type tuberculosis.

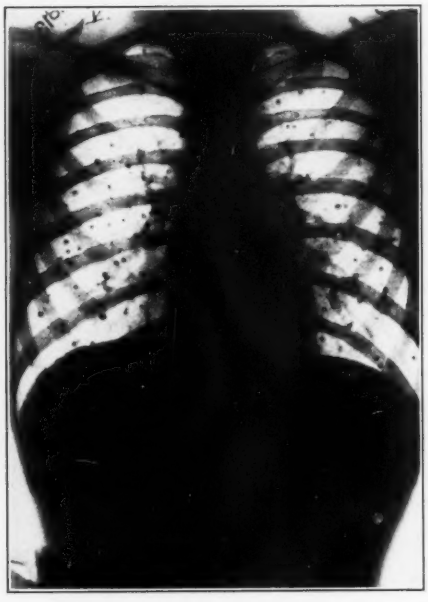

Fig. 5
Generalized tuberculosis of the lung.

hilum on the right side. The mother died of pulmonary tuberculosis. She gives a two-plus tuberculin reaction.

Case 5, Fig. 5.—D. J., a white girl, 8 years of age, shows generalized tuberculosis of the lungs, probably from a slow leakage through the tracheobronchial lymph nodes. She has a sister ten years of age who has a similar lesion in the lungs. Her father died of tuberculosis and her mother and two brothers now have tuberculosis in the active stage. She is 15 per cent underweight with no other symptoms. The tuberculin test is three-plus. These two children were found in a routine examination of tuberculin tests in the schools.

Case 6, Fig. 6.—B. C., a white child of 14 months who ran a temperature of 102 to 104° for two weeks. Physical signs were those of a general bronchitis. The baby lost steadily and vomited for three weeks, at the end of which time an x-ray of the chest was taken which showed miliary tuberculosis of the lung. The tuberculin test was three-plus. The baby was exposed to the grandmother who had complete charge of it. The baby died of miliary tuberculosis of the lung.

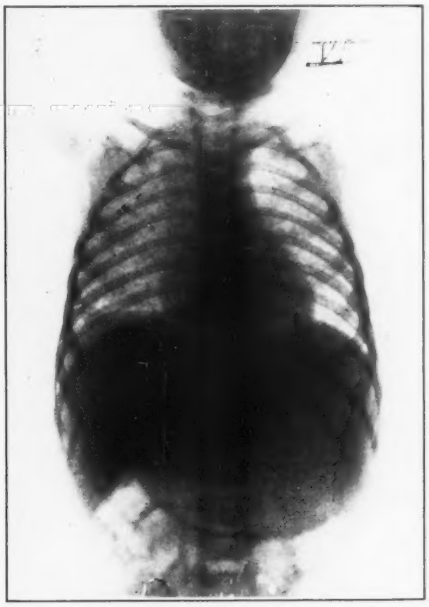

Fig. 6
Miliary tuberculosis of the lung.

Case 7, Figs. 7 and *8.*—M. B., an 8 months old white infant, normal delivery, nursed breast six months, at which time he developed whooping cough. For six weeks his temperature ran between 101 and 103°. Physical signs in the chest were those of a generalized bronchitis. The tuberculin reaction was two-plus. He was exposed to the cook, who had open pulmonary tuberculosis and had a pulmonary hemorrhage two weeks before the baby died. The cook took care of the baby on Wednes-

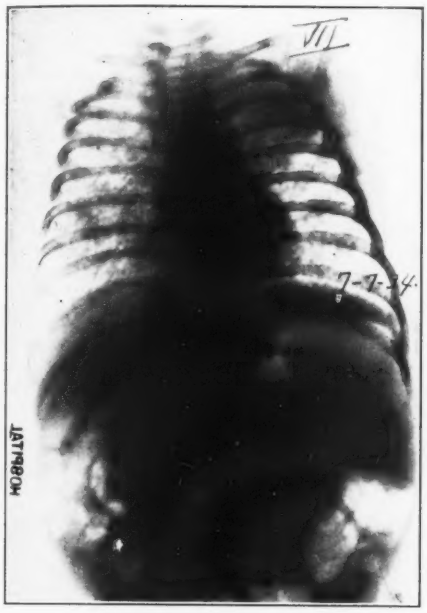

Fig. 7
Miliary tuberculosis of the lung.

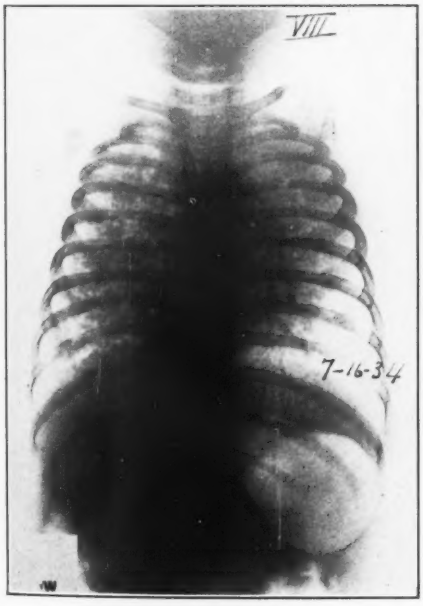

Fig. 8
Miliary tuberculosis of the lung.

day afternoons when the mother attended her card club. At all other times the mother had complete care of the baby. An x-ray of the chest showed miliary tuberculosis of the lung. The first plate was taken on July 7, 1934, and the second on July 16. The difference between the two plates shows the extensive spread which had occurred in one week. The patient died at nine months of age of generalized miliary tuberculosis of the lung.

Case 8, Fig. 9.—V. S., a white school girl of 11 years, who had adult type tuberculosis in the right lower lobe with a cavity in the right upper. Her sputum was positive for tubercle bacilli. Her father and mother both have active tuberculosis. A sister died of tuberculosis and pneumonia and a brother has active tuberculosis at the present time. Her weight is normal. She runs an afternoon temperature.

Fig. 9
Adult type tuberculosis.

Case 9, Fig. 10.—T. H., a well nourished baby of 12 months, was admitted to the hospital with a history of projectile vomiting for four days and temperature from 101 to 103° for one week. On account of the projectile vomiting a tentative diagnosis of brain tumor was made before admission to the hospital. On admission to the hospital the baby had a stiff neck, a positive Kernig, and a Babinsky. A spinal puncture was done which showed clear fluid under pressure, in which were found many tubercle bacilli. He was breast fed for six months, did well and his weight was normal.

After the diagnosis of the baby had been established, both the father and mother were examined. The mother's examination was entirely negative except that she

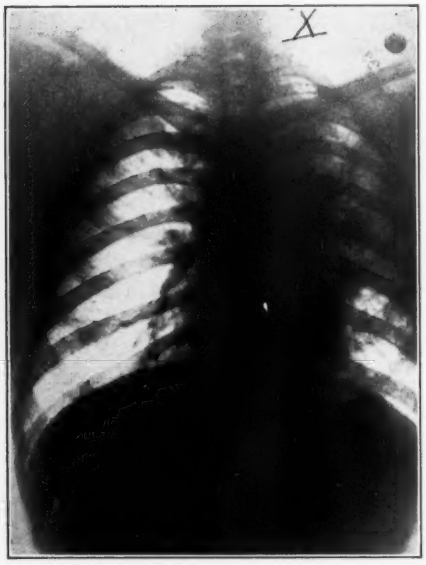

Fig. 10
Adult type tuberculosis.

was somewhat underweight. *Fig. 10* is the x-ray of the father, which shows definite involvement of his left upper lobe. Sputum of the father examined at that time showed many tubercle bacilli. He gave a history of having had influenza eight months previously and since then he had had a morning cough. The father was definitely the contact for the baby, was admitted to the North Carolina Sanatorium and was discharged cured in two years. The baby died of tuberculous meningitis.

Case 10, Fig. 11.—C. H., a colored boy 12 years old, shows childhood and adult type of tuberculosis. Large caseous lymph nodes are seen in the right hilum and tuberculous infiltration and thickened pleura at the right base. The tuberculin test is three-plus. The uncle with whom he lived died of tuberculosis.

Case 12.—R. H., a colored baby 17 months of age, was admitted to the hospital with diagnosis of whooping cough. The cough was quite severe and lasted for six weeks. During all this time his temperature ran each day from 101 to 105°. His general condition was very good. Physical signs showed only those of a bronchial pneumonia in the right chest in the region of the third and fourth interspaces. After three weeks an x-ray was taken of the chest, which showed a caseous lymph node at the right of the heart. This was at first thought to be bronchial pneumonia. Temperature and cough continued for four weeks longer and a second x-ray was taken, which showed miliary tuberculosis of the lung. The tuberculin test was three-plus. Examination of the gastric washing was negative for tubercle bacilli. Spinal fluid showed twenty cells. He continues to run temperature to 102° daily. His general condition is good in spite of the x-ray findings. The contact was not located.

Case 13.—R. G., a white infant 9 months of age, born of a tuberculous mother, did not nurse the breast. He was first seen by me in 1928 at the age of 9 months, when he weighed 24 pounds. He was very well nourished, slightly pale, and was brought for examination because of swelling of the right ring finger. On physical examination he showed consolidation of the right upper lobe. His temperature was 99°. He looked and felt very well. The tuberculin test was four-plus. The mother was treated at the North Carolina State Sanatorium before marriage and was advised not to marry. X-ray of the baby showed consolidation of the right upper lobe and dactylitis of the right ring finger. Due to these findings his parents were given a bad prognosis. Fortunately for him, four months later his mother succumbed to her infection. Since that time he has had repeated x-ray examinations of his chest which show no evidence whatever of the tuberculous lesion and only a small scar appears on his finger. He is now 9 years of age and weighs 74 pounds and is the picture of health. Because his contact with his mother was abruptly broken before he became overwhelmed with his infection this boy is healthy today.

RECOMMENDATIONS

(1) All school children should be given the tuberculin test.

(2) All nurses, maids and cooks should be given an x-ray examination of the chest.

(3) All school teachers should be given the tuberculin test and those positive should have x-rays of the chest.

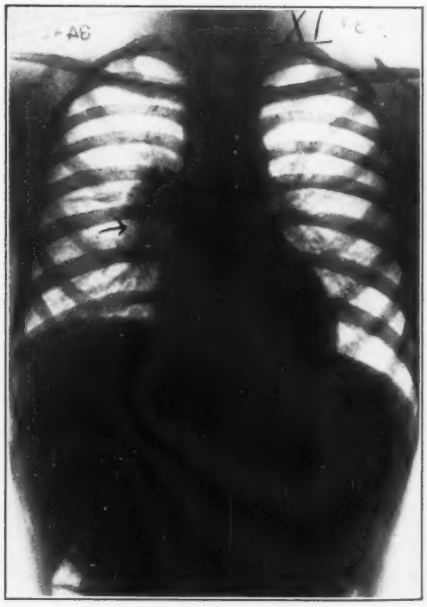

Fig. 11
Childhood and adult type tuberculosis.

To quote from Charles Hendee Smith:[6]

"It must be remembered that 'the army of the tuberculous is recruited from those infected in childhood,' and 'Phthisis is the last verse of a song the first of which was sung in the cradle.' These infected children, then, who carry tubercle bacilli in their bronchial lymph nodes or elsewhere, are the ones who need early, correct diagnosis. The Mantoux test provides the means of making it. It should be used on every child who is underweight, anemic or languid, or who has an unexplained irregular fever. These children with 'tuberculous infection' have true incipient tuberculosis, latent or active. Their disease is generally in the lymph nodes, where it may be encapsulated and where scar tissue does no great harm. Under intelligent, watchful care they usually do well. When the disease has involved more important structures, such as the lungs, bones, or meninges, the damage is greater, the cure is more difficult or impossible, and the diagnosis has been made too late."

TREATMENT

It may be said in general that the treatment is essentially the same as that of adult tuberculosis. Each patient should be studied closely to try to locate the contact and see that there is no further infection.

The childhood type tuberculosis as a rule needs no specific treatment. If these cases have no symptoms and no fever, as is the usual story, they are allowed to follow the routine of a normal, healthy child of that age except to avoid too strenuous exercise. A rest period each afternoon is prescribed.

Cases which have primary lesions in the inflammatory stage or caseous glands and fever are kept in bed and given fresh air and plenty of nourishing food.

Cases that have the adult type of tuberculosis are treated as one would the same lesion in the adult. In case of a cavity, pneumothorax therapy is given. The advanced lesions are treated just as in adult tuberculosis.

What we need most of all is a preventorium where these children with pulmonary lesions whose home conditions are unfavorable may be properly treated. In most states no provision is made for the care of tuberculous children. They are not allowed admission to the tuberculous institutions until they are 5 or 6 years of age or older. Often infants and young children have to continue to live in intimate contact with a tuberculous mother or other relative. Such overwhelming infection is responsible for many infant deaths. Treatment at this age can accomplish so much more than at any other period of the child's life.

BIBLIOGRAPHY

1. Chadwick, H. D.: Mich. State Med. Society, **31**:109-112 (Feb.) 1932.
2. Zachs, David: New England Med. Jour., Sept. 10, 1931.
3. Myers, J. A.: Tubercle, Feb., 1933.
4. Chadwick, H. D.; and McPhedran, F. M.: Childhood Type of Tuberculosis.
5. Morse, J. L.: Boston Med. & Surg. Jour., **173**:654-656 (Oct. 28) 1915.
6. Smith, Charles Hendee: Amer. Jour. Dis. Child., **38**:1137-1154. McPhedran, F. M.: The Amer. Rev. of Tb., **20**:4 (Oct.) 1929.
7. McPhedran, F. M.: The Canad. Public Health Jour., Oct., 1930.
8. Merritt, Katharine: Amer. Jour. Dis. Child., **38**:526-540 (Sept.) 1929.
   Smith, Charles Hendee: Ann. Int. Med., **8**:9, 1935.
9. Alley, Leon A.: New England Jour. Med., **207**:884-892 (Nov. 17) 1932.
10. Von Reuss: The Diseases of the New Born. Wm. Wood & Co., 1921, p. 626.
11. Whitman, R. C.; and Green, L. W.: Arch. Int. Med., **29**: 262 (Feb.) 1922.
12. Hill, Lee Forest: The Journal-Lancet, **56**:183-187 (April) 1936.
13. Lincoln, E. M., Bellevue Hospital, New York City.
14. McCain, P. P.: Personal communications.
15. Merritt, Katherine: Amer. Jour. Dis. Child., **38**:526 (Sept.) 1929.
16. McLean, C. C.: Sou. Med. Jour., p. 204, March, 1928.

DISCUSSION (*Abstract*)

Dr. Miriam E. Brailey, Baltimore, Md.—I have listened to Dr. Sidbury's discussion of tuberculosis in childhood with great interest and have sympathized especially with the emphasis he has placed on the necessity of using tuberculin and x-ray in diagnosing tuberculosis in children. The least important contribution to exact knowledge about the young child's true tuberculous status will be made by the ordinary physical examination unless he is an extremely ill case with a very poor prognosis. In our experience, which has been confined mainly to infants and children of the preschool group, impaired nutrition and fever are late accompaniments of extensive disease, and in children under 5 years of age their absence may seriously mislead the clinician.

It is clear, I think, that infected infants, that is, children under 2 years of age, show lesions in a higher proportion of cases than the run of tuberculin-positive children between 5 and 15 years of age. In the Harriet Lane Tuberculosis Clinic, among 170 babies found to be Mantoux-positive before 2 years of age, 67, or 39 per cent, were shown by x-ray to have parenchymal involvement usually accompanied of course by enlarged tracheobronchial nodes. There were 36 others who on initial x-ray examination showed definitely bulging tracheobronchial nodes without evidence of lung involvement. If these are included, the proportion with lesion when first seen after the discovery of a positive tuberculin rises to 61 per cent. Subsequent observation of these children by serial x-ray has shown these readings to be correct as judged by the duration of consolidations and the eventual deposition of hilar calcium in a high proportion of survivors observed as long as 3 years.

With reference to these 170 tuberculin-positive infants who probably represent the most susceptible age group, it is of interest to note that only 22 were actually ill when first seen. This is 13 per cent of the whole number. In these cases diagnosis could usually have been approximated fairly closely by an experienced clinician without the aid of tuberculin and x-ray. In the remaining 148 (87 per cent of the total) the children were in apparent good health and physical examinations done

by doctors looking for tuberculosis were negative. Their diagnosis could in no case have been made when it was without the aid of tuberculin and x-ray. Yet among these 148 lay concealed 45 pulmonary cases, many of them showing x-ray shadows nearly as extensive as those displayed by the 22 sick ones first mentioned.

In this group of 170, 72 of whom were white and 98 colored, we found a mortality of about 10 per cent for white infants during the first year of clinical observation; during the same time period colored infants of the same age suffered a mortality of 21 per cent. In a five-year period of observation, the white death rate rose only to 13 per cent, and the colored to 31 per cent. Thus for both races 70 per cent of all the fatalities occurring over a five-year period of observation occurred within the first twelve months after we found the tuberculin test positive. This suggests that for the preschool child the first year of infection is where the main hazard lies, and that after that, vigilance can be relaxed if recession of the lesion is taking place or if nothing definite has appeared in the chest plate by that time.

It is well recognized that in children under 5 years of age nearly 75 per cent of the fatalities from tuberculosis are due to extrapulmonary forms of the disease notably to miliary tuberculosis and to tuberculous meningitis. Although such fatal complications may occur with receding or sometimes apparently insignificant chest lesions this is fairly rare and the chest plate remains our best objective measure of the child's status.

It seems especially regrettable that the state sanatorium system in most states makes no provision for the care of the tuberculous infant and preschool child. Children in Maryland must be 6 years of age to be eligible for state sanatorium care and only ambulatory cases are welcomed. The age group from 5 to 10 is the one least in need of sanatorium care, and those who do need it are not ambulatory cases. Those who should get sanatorium care are the very ones who at present receive the minimum attention in tuberculosis surveys and in so-called preventive work; the preschool child and the adolescent in our high schools. We could profitably dispense with most of our mass interest in the grade school child, so far as tuberculosis is concerned, and instead concentrate on children of any age who come from homes with open tuberculosis in some adult. Even then the children most gravely in need of clinical care and at least annual x-ray of the chest are children under 5 and those over 12. For the latter group in the white race at least, there already exist public institutions for their care if minimal or progressive disease is discovered. For the young child, even if it be white, there is no such provision outside of our hospitals for acute disease.

For the colored of any age where the mortality from tuberculosis for the race as a whole is in many Southern states four times that for the white, a whole book could be written on their need and our failure to meet it adequately.

Dr. P. P. McCain, Sanatorium, N. C.—It used to be thought that all infants with positive tuberculin tests would die of tuberculosis. We now know that not only is this not true, but that a large proportion of the infants and children showing definite tuberculous lesions by x-ray will get well under proper treatment if they are prevented from receiving further infection. This is especially true of children who have lesions of the primary type. Many children with adult type tuberculous lesions are also being cured by artificial pneumothorax.

Dr. Sidbury has properly emphasized the fact that the diagnosis of tuberculosis in infants and children depends almost entirely upon a positive tuberculin test and properly taken x-ray films. He has also properly emphasized the great need of making a study of all children for tuberculosis, for we never know that they have not been exposed to tuberculosis from some servant or other source, even though every member of the family is known to be free from tuberculosis. Public health and anti-tuberculosis workers often find that through a study of groups of apparently healthy children and by a study of the contacts of the positive reactors, and especially of the positive reactors showing demonstrable lesions, they can find many new cases of tuberculosis among adults.

The most important step in the treatment of children having any form of tuberculous infection or childhood type tuberculosis is to see that they do not get any further infection. To do this it is necessary to locate the source of the child's infection and to break the contact. The positive sputum case who is infecting the child should be sent to a sanatorium and kept there until he learns how to practice all the necessary precautions to protect others in the home. If this cannot be done, the child should be sent to a relative or to a preventorium or children's sanatorium.

If we prevent that large group of children who have positive tuberculin tests, but no demonstrable lesions, from being further infected with tubercle bacilli, we shall rob the Great White Plague of many of its future victims. If the resistance of these children having positive tuberculin tests but no demonstrable lesions is built up through the correction of defects, good food, regular habits, *et cetera,* they can safely be allowed to live like other normal children. As a matter of precaution x ray films of their chests should be made every two or three years. With proper care and reasonable supervision practically none of these children should ever develop clinical tuberculosis.

Those children having demonstrable childhood type lesions which are calcified, by far the most common type of lesion found in children, should have the same good general care as the group above mentioned, but should in addition avoid strenuous games, such as football, and should lie down for an hour or two in the afternoon. Practically all of them can safely go to school and they should have x-ray films made every year. If they do not get further infection and if they get reasonably good care, practically none of them should develop the adult type of tuberculosis.

Those children having large tracheobronchial glands in the inflammatory or caseous stage, and those having childhood type lesions with primary foci in the inflammatory stage, in my judgment should have bed rest in addition to good general care and freedom from further infection until x-ray films show a very definite improvement in the lesions. With proper care the prognosis in these cases is also good.

Children having the adult type of tuberculosis should have the same sort of treatment as adults, including pneumothorax when this is indicated. The outlook in this type of cases is not so unfavorable as it was once supposed to be. Practically all early stage cases and many more advanced cases respond favorably to treatment.

TRANSPLANTATION OF THE HUMAN CORNEA*

PRELIMINARY REPORT

By J. WESLEY MCKINNEY, M.D.
Memphis, Tennessee

The restoration of vision which has been lost from opacities in the transparent media has been the concern of ophthalmologists from time immemorial. Until recently loss of vision from corneal opacity has been hopeless.

Opacity of the lens has attracted the attention of all medical men as well as those especially interested in the eye. In 1753, Daviel presented his method of cataract extraction and ever since the surgery of cataract has progressed with modifications and improvements of technic until nowadays cataract extraction is a successful procedure in a high percentage of cases.

The visual results in corneal surgery cannot now, and probably never will, except in rare incidences, compare with those obtained after uncomplicated cataract extraction. This is easy to understand when we consider the injury beyond the cornea that is usually associated with leukoma. If, however, we can improve a blind person's vision to a degree that will enable him to get around without assistance, we shall have done him a great service.

The first unsuccessful and even disastrous attempts at corneal transplantation, which have only a historical value, were followed by others that gave more hope. The recent statistics of Elschnig, Filatov, Thomas and Castroviejo are truly remarkable. The idea of a transplant from the cornea of one human being to another seems almost fantastic, yet at the present time it is being done with a surprising number of successes.

It is interesting to review the older attempts at substituting transparent tissue for the opaque cornea.

In 1789 deQuengsy proposed the substitution of glass for opacified corneae. Several attempts at this were made, but were, of course, unsuccessful.

Wagenmann, in 1888, was the first to describe transplantation of the whole cornea with a portion of the conjunctiva attached.

The first case reported in the literature was presented by Filatov in 1913. He transplanted the whole cornea together with a small margin of sclera and conjunctiva. In the same year Schimanovsky reported two cases in which the whole anterior segment of the eye, with the exception of the iris, ciliary body, lens, was transplanted.

Several other attempts at total keratoplasty were made, notably by Burke in 1921, Elschnig in 1922, and Key in 1930.

All of these transplants became opaque and in most of them glaucoma and phthisis bulbi ensued.

Von Walther suggested that the best results in keratoplasty might be obtained when only the superficial layers of the cornea were transplanted, that is, a lamellar keratoplasty.

Following the idea of von Walther, Mülbauer (*Fig. 1*), in 1840, attempted transplantation of the superficial layers of the cornea of animals to human beings, heterotransplantation. After the transplants healed, leukomas formed, some of them prominent and bulging. This quenched the enthusiasm of the author and other workers of the time. Again in 1877, attempts at heterotransplantation met with the same results.

In 1888, von Hippel (*Fig. 2*) presented his

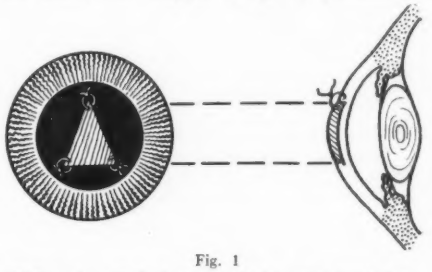

Fig. 1
Mulbauer's method of circumscribed lamellar keratoplasty.

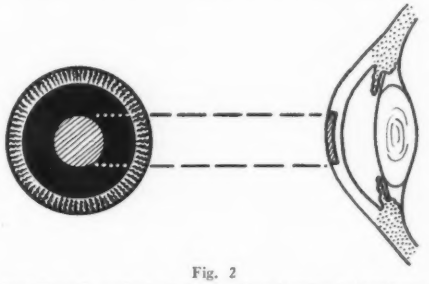

Fig. 2
Von Hippel's method of circumscribed lamellar keratoplasty.

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method of circumscribed lamellar keratoplasty. With his trephine he removed discs of part thickness of the cornea and four to five millimeters in diameter and replaced them with discs of the same diameter, but of full thickness. He stated that a certain degree of transparency of the transplant was obtained. Von Hippel, who had also done penetrating keratoplasties, concluded that the lamellar keratoplasty was preferable because it was easier to perform and less dangerous.

Fuchs reported 30 operations performed after von Hippel's technic and saw improvement in only two.

Löhlein (*Fig. 3*) presented an operation which he first tried on rabbits and then on human beings. Using parallel knives, he outlined a strip 5 mm. wide from the upper to the lower limbus. The strip of leukomatous cornea was dissected off without penetrating and replaced by a strip dissected in a similar manner from the eye of a living human being or one just after death. He was able to obtain a vision of 6/60 in one case.

Several others reported operations after the technic of Löhlein, but most of the transplants became opaque.

Morax (*Fig. 4*), in 1912, interchanged a lamellar transplant cut out with a trephine from the clear periphery of the cornea with a similar one from the opaque center of the cornea. Several others attempted varieties of this procedure without much success.

Wiener (*Fig. 5*) introduced a variation in 1913, and still performs a lamellar keratoplasty without a transplant. He makes a crucial incision in the scar and, using a sharp hook to the corner of each of the four flaps, dissects them off the cornea and allows epithelialization to take place. Some improvement in vision is obtained.

In all these cases of lamellar keratoplasty, even though all the scar was removed and even if the graft remained clear, there was scarring at the plane of the dissected cornea which is comparable to that seen after the removal of a pterygium.

The first attempts at transplanting a portion of the full thickness of the cornea was made by Reisinger in 1824. He worked on rabbits, but was unable to obtain transparent grafts.

Wolf, in 1873, devised the operation later described by Löhlein except that he used the penetrating type of keratoplasty rather than the lamellar type.

In 1877, von Hippel (*Fig. 6*) described his penetrating keratoplasty, using the trephine to

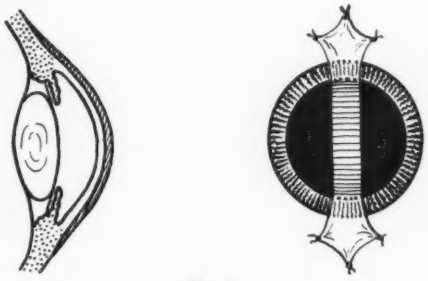

Fig. 3
Lohlein's method of circumscribed lamellar keratoplasty.

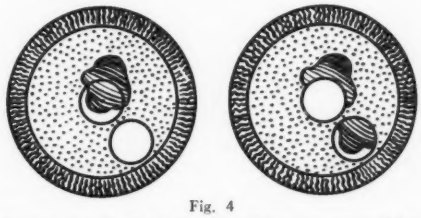

Fig. 4
Morax's method of lamellar keratoplasty by transposition.

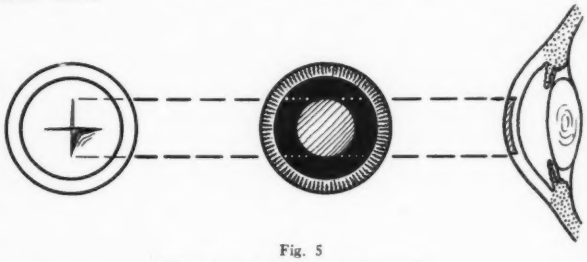

Fig. 5
Wiener's method for corneal resection.

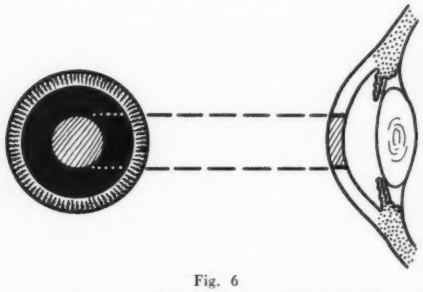

Fig. 6
Von Hippel's technic of circumscribed penetrating keratoplasty.

obtain transplants which would be the same shape as the defect. The transplant was held in place by pressure of the lids only.

Sellerbeck, in 1877, used the von Hippel technic and added the conjunctival flap dissected from below and sutured to the upper limbus to hold the graft in place. He reported no successful cases.

Zirm (*Fig. 7*), in 1906, reported the first real success in penetrating keratoplasty. He operated by von Hippel's technic, with the addition that the graft was held in position by cross sutures inserted into the conjunctiva close to the limbus. He was able to obtain a vision of 6/36 in a case of leukoma following a lime burn.

In 1914, 1916 and 1931 Leoz Ortin conducted experiments that were particularly interesting in the study of nerve regeneration in the transplants. He cut out plugs of varying shapes and sutured them back into place and transplanted plugs from one rabbit to another. By special staining he found that the corneal nerves had regenerated. It was his impression that one of the most important factors in the success of the transplant was coaptation of the edges rather than of surfaces and that nervous regeneration in the graft was necessary for its permanent transparency.

In 1921, Ebeling and Carrel (*Fig. 8*) made a rectangular graft with a step in its edges and used sutures into the graft to hold it in place. Of five cats operated upon only one transplant maintained any transparency.

The first real series of cases was reported by Elschnig (*Fig. 9*) in 1930. He reported upon 174 cases operated upon in his clinic during the preceding twenty years. He used the technic of von Hippel, but held the transplant in place by means of a bridged suture from the upper to the lower limbus. He used eserine to constrict the pupil so that the lens might not be injured by the trephine. Elschnig arrived at the conclusion that the circumscribed penetrating keratoplasty of von Hippel was the only dependable method; that grafts might be taken from the eyes of living persons, young or old, whether the remaining part of the anterior segment of the eye were normal or pathologic, glaucomatous or hypotensive or from the eye of an adult or infant shortly after death. He did not find any relationship between hemolysis or agglutination of the blood and viability and transparency of the graft. In his best case he obtained a vision of 6/6.

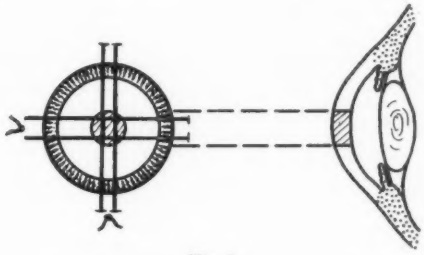

Fig. 7
Zirm's technic to hold the transplant in position.

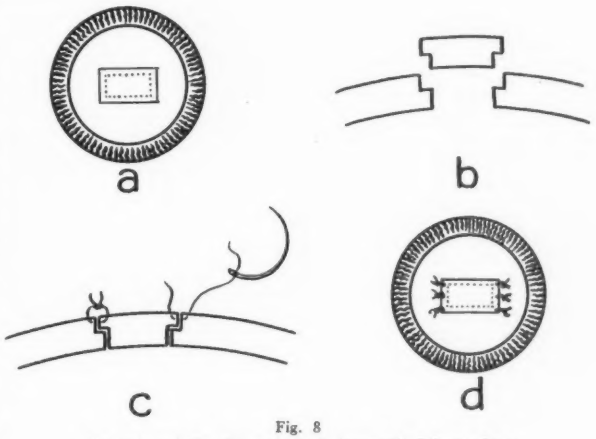

Fig. 8
Ebeling's and Carrel's method of circumscribed keratoplasty.

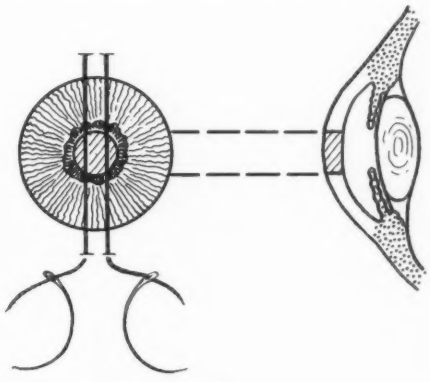

Fig. 9
Elschnig's technic to keep the transplant in position in von Hippel's circumscribed keratoplasty.

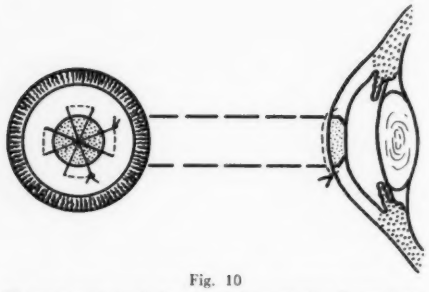

Fig. 10
Thomas's technic of circumscribed penetrating keratoplasty.

Thomas (*Fig. 10*), in 1930, performed a series of experiments on rabbits, using transplants of different shapes and operating by different technics. He came to the following conclusions: (1) that forceps should not be used to handle the transplants; (2) that transplants should be beveled, because this increases the surface of contact favoring nutrition and cicatrization of the graft; (3) that the graft should be round, because the corners have a tendency to protrude and cause detachment of the graft; (4) that sutures in the graft traumatize it and produce lines of uneven tension which may cause detachment of the graft and fistula formation; (5) that when the iris becomes adherent the transplant is invaded by blood vessels and becomes opaque; (6) that when the transplant is the same size as the defect the transplant swells and does not fit, therefore a slightly smaller trephine should be used to obtain the transplant; (7) irregularities of 1 mm. resulting from technic are of fatal consequence; (8) that the pupil should be dilated to the maximal extent to prevent anterior synechiae and finally that the round transplant cut out with von Hippel's trephine and scissors with beveled edges and crossed stitching give the best results.

Filatov (*Fig. 11*) has modified the von Hippel technic by using a strip-like conjunctival flap dissected from above and turned down over the graft with raw surfaces outward and sutured to the conjunctiva at the lower limbus. He further modified the technic of von Hippel by making a puncture and counter puncture about 8 mm. apart with the cataract knife, thus making parallel perforating wounds in the cornea. A strip of celluloid was pressed through these wounds, separating the cornea from the iris and lens. The cornea was then trephined with this strip in position acting as a protection to iris and lens and preventing loss of vitreous in an aphakic eye. He reported 94 cases operated upon by this method. His best visual result was 30/60.

Castroviejo operated upon a series of over a hundred rabbits by various technics and finally developed a technic of his own which he considered superior to any other. At first he cut out a rectangular flap 6 mm. long and 4 mm. wide, using a special twin knife, keratome and scissors that he developed. Later he used a flap 4 mm. square. He made sections to show that the edges of the flaps cut out with a trephine were much more ragged than those cut out by his method. He has recently reported a very important series of cases done on human beings by his technic. He was able to obtain a vision of 20/30 in his best case.

This brief historical review of the development of corneal transplantation during the past hundred years serves to focus our attention on this most interesting subject.

Realizing from the start that it would require much work to perfect my own technic and to learn how to overcome the most unusual difficulties that the conditions associated with corneal leukoma present, and with the hope that I might contribute something to the solution of the problem, I began about ten months ago to work on rabbits. During these ten months I have operated upon about 45 rabbits. Unfortunately, rabbits do not seem to be very good material for this type of operation, because many of them become infected, and because they cannot be

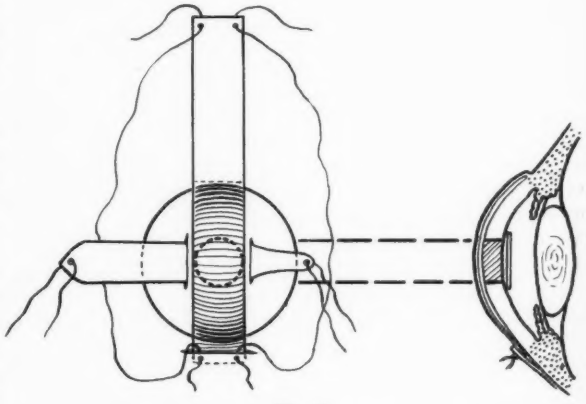

Fig. 11
Filatov's technic of circumscribed penetrating keratoplasty.

kept still or prevented from clawing at the eye after operation. They are, however, easy to handle and offer a satisfactory proving ground for corneal surgery.

At the onset of the work with rabbits I used the various modifications of von Hippel's technic and the latest technic of Castroviejo. At the present time I consider Castroviejo's technic the best to be advanced so far and shall present it in detail later on in this paper. I have attempted to reproduce in rabbits' eyes many of the conditions associated with corneal leukoma, such as anterior synechiae, aphakia, ectasia, and vascularization. This phase of the work has been very instructive and I shall report on it in a later paper.

What are the cases that are suitable for corneal transplantation? In general, it might be said that all cases of corneal leukoma in which the opacity is too extensive for optical iridectomy and in which there is a fair degree of light projection are candidates for corneal transplantation. Those cases in which the anterior chamber is reasonably normal as determined by anterior and posterior transillumination and in which some corneal elements remain offer the best prognosis. This situation might obtain as a result of interstitial keratitis, trachoma, and ulcers which have not perforated. Less favorable are those cases of leukoma in which there are total dense opacity without corneal elements, anterior synechiae, ectasia, staphyloma and aphakia. Many of the unfavorable conditions may be eliminated before transplantation. A preliminary transplant, either penetrating or lamellar, may be done to supply the corneal elements for the final graft. Anterior synechiae may be divided. Intra-ocular pressure may be controlled by operation. Staphylomas may be excised and the superficial vessels of extensive pannus may be obliterated by surgical diathermy. Contraindications to corneal transplantation are glaucoma which cannot be relieved by operation, active iridocyclitis and absence of light projection. Faulty light projection is no contraindication as the retina is often found sufficiently normal after the operation.

From what sources is the material for corneal transplantation obtained? There are two sources of material, namely, autogenous and homogeneous.

It sometimes happens that the other eye of a patient with corneal leukoma is blind, but has a usable cornea. In this case a graft is taken from this eye and transplanted to the leukomatous eye. Rather than remove the donor eye, then, the defect is filled in with fascia lata. A situation like this, in which the autotransplant might be used, rarely presents itself. Consequently, homotransplantation is the usual procedure. The material is obtained from the eye enucleated from a living human being because of trauma, intra-ocular new growth, absolute glaucoma, and so on. The eye may be obtained from the body at autopsy up to ten hours after death. This has proven to be just as good material as that taken from the eye of a living person. On removal of the donor eye it is placed in sterile normal saline until ready for use. It may be used immediately or kept in a refrigerator at 2 to 6° C. for three days and still be in perfect condition for transplantation. There seems to be only one condition that the donor of an eye, whether living or dead, must fulfill, that is, that he must not have any general or local infection. Grafts have been transplanted with equal success whether obtained from the six-months-old fetus or the old man. Blood compatibility is of no importance.

The operation (*Fig. 12*) that I have used follows, with a few minor modifications of my own, the most recent technic of Castroviejo. Atropine is instilled several times the night before and the morning of the operation.

On the table, 4 per cent cocain is instilled. Akinesia is obtained by the Van Lint method. Retrobulbar injection of 2 per cent procaine hydrochloride with 2 drops of epinephrine is done. A four point injection of epinephrine at the limbus is made to assure wide dilation of the pupil. Sutures are put into the lids as retractors as no pressure must be made on the eyeball. A bridle suture is put around the superior rectus and sometimes the inferior rectus as well. The

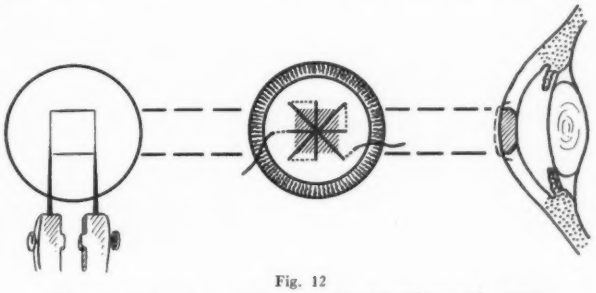

Fig. 12
Castroviejo's technic of partial penetrating keratoplasty with sutures.

eye is fixed with an Elschnig forcep and a section 4 mm. square is outlined in the cornea by means of a twin knife at the site previously determined. Then sutures are put into the cornea about 1 mm. from the section all around to form a sort of maltese cross effect. To do this the needle enters the superficial layers of the cornea and traversing about 2.5 mm. of cornea comes out at the lower right corner, enters at the upper left corner and comes out at the middle of the upper side, enters at the middle of the lower side and comes out at the lower left corner, enters at the upper right corner and comes out at the middle of the right side, enters at the middle of the left side and comes out 2 mm. beyond. This single suture is left in large loops and smoothed out of the way while the section is being cut out. A traction suture is then put into the square section. The square is then outlined by instilling fluorescin. A keratome incision at an angle of 45° is made at the lower side (nearest the operator) of the square and the rest of the square is cut out with special scissors, while traction is being made with the suture. This then gives a square defect, the margins of which are leveled about 45°. The traction suture, by pulling the square away while it is being cut, prevents injury to the lens. At this time anterior synechiae are freed with a spatula. If the lens is seen to be cataractous it may be removed now or later. The recipient eye having been prepared, the donor eye is wrapped in gauze, leaving the cornea exposed. The graft is then cut out in exactly the same manner as in the recipient eye with the exception that no traction suture is used. The graft is then transferred from donor to recipient by a spatula without trauma and the sutures drawn taut and ends tied. All lid and bridle sutures are removed and lids sealed with a thin piece of wet cotton.

The patient remains in bed for ten days. The unoperated eye is uncovered on the fifth day. Sutures are removed on the seventh day, and the patient goes home two weeks after the operation.

CLASSIFICATION OF RESULTS

Thomas, of London, has presented a classification of results which should be universally adopted. The classification is as follows:

Successes:

(1) Clinically transparent graft.

(2) Slight degree of opacity in or behind graft.

(3) Much opacity in graft, but less than original opacity.

Failures:

(1) Much opacity, that is, equal to or greater than original opacity.

(2) Graft detached.

(3) Eye lost.

Thomas reviewed the results of his own cases and those of Elschnig, Filatov and Castroviejo in the light of this classification. In all there were 338 operations on favorable and unfavorable cases. Of these 216 were favorable cases in which successes were reported by Elschnig, in 46 per cent; by Filatov in 45 per cent; by Thomas in 83 per cent; and by Castroviejo in 81 per cent. The cases of Elschnig and Filatov extend over a much earlier period than those of Thomas and Castroviejo. In the unfavorable cases 38 per cent of successes were reported. It might now be conservatively stated that with suitable technic success may be expected in 3 out of 4 of the favorable cases and 1 out of 3 of the unfavorable cases.

REPORT OF CASES

Case 1.—M. W., aged 37, had a corneal leukoma of the right eye as a result of repeated ulcers as a child. The cornea was somewhat ectatic with a small relatively clear area just below the upper limbus. The iris was adherent below. The tension was normal. Vision was equal to counting fingers at 3 feet with good light projection. The left eye also showed leucoma with a vision of 6/60. Keratoplasty was performed January 12, 1937.

Postoperative Course.—The graft was clear at the first dressing. On about the tenth day a slight subepithelial haze developed above, associated with some deep vascularization. After about 6 weeks a few microscopic vesicles developed in the epithelium of the graft and surrounding cornea. These cleared up after about three weeks. On the whole, the graft has remained quite clear. The vision is 4/200.

Case 2.—O. W., aged 25, had a dense leukoma of the left eye in which iris was everywhere incarcerated as

Fig. 13

Photograph of Case 1 (left) and Case 2 (right) taken two months after operation. The central square is the transplant surrounded by scarred cornea.

a result of a firecracker burn five years previously. Vision was hand motion against a strong light. Light projection was good. The right eye was normal, having a vision of 6/4 5. Keratoplasty was done January 12, 1937.

Postoperative Course.—The graft was clear at the first dressing and has remained clear since. There has been no vascularization of the graft. There has been a microscopic vesiculation of the epithelium on, and immediately surrounding the graft. The vision is 10/200.

Donor.—The donor eye for both these cases was enucleated at the time of the transplantation because of carcinoma at the limbus. There remained enough normal cornea to furnish two grafts.

COMMENT

Both of these cases were unfavorable for corneal transplantation. In the first case the cornea was ectatic and the iris was widely incarcerated, two unfavorable conditions. At operation an anterior polar cataract could be seen and later with the slit lamp posterior cortical lens changes could be seen. In the second case there was dense scarring in which the iris was everywhere bound. At operation anterior polar cataract was found and later posterior cortical lens changes were noted with the slit lamp. It is planned to remove the cataracts at a later date.

SUMMARY AND CONCLUSIONS

It has been the purpose of this paper to review the development of corneal transplantation over the past one hundred years and to show that now keratoplasty can be successfully performed in a large percentage of cases.

Rabbits offer a satisfactory proving ground for the exacting technic necessary for corneal surgery.

Until the advent of successful keratoplasty a person who was the victim of an injury, burn or disease which caused any extensive opacity of the cornea was doomed to blindness for the rest of his life. Now he may hope for the restoration of useful, if not perfect vision.

Favorable cases are those in which the anterior chamber is reasonably normal and in which the corneal opacity is neither so extensive nor so dense that no corneal elements remain. Less favorable are those cases which have total dense corneal opacity without corneal elements, anterior synechiae, ectasia, and staphyloma. Many of the unfavorable conditions may be eliminated before transplantation.

If a suitable technic is used we may expect a successful grafting in 3 out of 4 of the favorable cases and in 1 out of 3 of the unfavorable cases.

The source of material for corneal transplants is either autogenous or homogeneous. Heterogeneous grafts have been uniformly unsuccessful.

BIBLIOGRAPHY

Burke, J. W.: Total Keratoplasty. Arch. of Ophth., **50**:147, 1921.

Castroviejo, R.: Keratoplasty, a Historical and Experimental Study, Including a New Method. Two parts. Amer. Jour. Ophth., **15**:825 and 905, 1932.

Castroviejo, R.: Keratoplasty, a Report of Seven Cases. Amer. Jour. Ophth., **17**:1932, 1934.

Ebeling, A. H.; and Carrel, Alexis: Remote Results of Complete Homotransplantation of the Cornea. Jour. Exp. Med., **34**: 435 (Nov. 21) 1921.

Elschnig, A.: Keratoplasty. Arch. of Ophth., **4**:165, 1930.

Filatov, W.: Transplantation of the Cornea. Arch. of Ophth., **13**:321, 1935.

Fuchs, E.: Quoted by Herman Kuhnt in Elschnig's Augenarztliche Operationslehre; p. 519. Berlin: Julius Springer, 1922.

Von Hippel: Eine neue Methode der Hornhauttransplantation. Arch. f. Ophth., **34**:108, 1888.

Key, B. W.: Report of a Case of Transplantation of the Human Cornea. Amer. Ophth. Soc., **28**:29, 1930.

Leoz Ortin, G.: Sobre practica y biologie de las plastias. Med. Ibera, **20**:213, 1931.

Lohlein, W.: Eine erfolgreiche methode der Hornhauttransplantation. Arch. f. Augenh., **67**:398, 1910.

Morax, V.: Kerotoplastic par transposition. Clin. Ophth., **19**: 108, 1912.

deQuengsy, P.: Quoted by Leoz Ortin.

Reisinger: Quoted by Herman Kuhnt in Elschnig's Augenarztliche Operationslehre; p. 517. Berlin: Julius Springer, 1922.

Schimanovski, A. T.: Transplantation des vorderen bulbsabschnittes. Klin. Monatsbl. f. Augenh., **51**:870, 1913.

Sellerbeck: Quoted by Herman Kuhnt in Elchnig's Augenarztliche Operationslehre, p. 518: Berlin: Julius Springer, 1922.

Thomas, J. W. T.:Successful Grafting of the Cornea in Rabbits. Lancet, **1**:35 (Feb. 7) 1931.

Thomas, J. W. T.: Brit. Med. Jour., Jan. 16, 1937.

Wagenmann, A.: Experimentelle Untersuchungen zur Frage der Keratoplastik. Arch. f. Ophth., **34**:211, 1888.

Wiener, M.: Surgical Removal of Corneal Opacities, Contribution to Ophthalmic Science. Edward Jackson Birthday Volume, p. 193. Menasha, Wis.: Banta Publishing Co., 1926.

Zirm, E.: Eine erfolgreiche totale Keratoplastik. Arch. f. Ophth., **64**:580, 1906.

CHRONIC MIDDLE EAR AND MASTOID INFECTION*

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When we are called upon to launch an offensive against an entrenched enemy it is of the utmost importance to make a careful study of his habits. Before we can make an intelligent plan it is necessary to know how strongly he is fortified. It is necessary to know his base of supplies and where it is situated. A frontal attack should never be made if there is a possibility of coming upon the enemy from the rear. We should attempt first to capture his base.

When we are confronted with a chronic puru-

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lent otitis media there is one thing that should be uppermost in our minds. A middle ear infection is a secondary infection, just as truly as tuberculous laryngitis is secondary. Remembering this we should always look for the primary focus in its accustomed place. I believe the primary focus is practically always in an infected sinus. At the time we are called on to examine the chronic purulent otitis the primary focus in the sinus may have temporarily or permanently cleared up so that we are unable to connect the original criminal with the crime under investigation. It may be that the present base of supplies lies in an infected mastoid cell. It may be that the criminal is being given aid and comfort by infected adenoids and tonsils. The most fortunate discovery is that the enemy has dug himself in within the cavity of the middle ear itself, for it is in this situation that his destruction is most easily accomplished.

We are now prepared to draw up a plan of campaign. The first and a most important step is the history. We inquire carefully how long the discharge has been noted. Did it make its appearance following an illness? If so what was the nature of the illness? We most frequently find that the discharge followed an attack of scarlet fever, measles, pneumonia, influenza or diphtheria. Oftentimes the history of the discharge goes back into infancy or early childhood and the patient is unable to give us any history of an illness. In that case we have to assume that the discharge followed one of the exanthematous diseases. Oftentimes the patient attributes the discharge to an acute cold. Those cases we are inclined to classify as an acute exacerbation of a chronic sinus infection. There are many cases following a plunge in a swimming pool or open water. It will generally be found that chronic cases with this history also have a chronic infection of a sinus or sinuses. What happened was that the patient flooded the opening of his eustachian tube with some of the pus from his own sinuses. Then there are the nasal douche cases which are strictly analogous to the swimming cases. They wash the pus from their own infected sinuses into the eustachian tube. Likewise the cases following nasal packing are caused by a transfer of the infected material to the tube. In the course of our inquiry we may find that the discharge came on without any cause so far as the patient could tell. It was preceded by no pain. The first thing the patient discovered was the discharge running from the ear. In this case we should suspect tuberculosis and a most careful examination should be made with that in view.

We have now covered the history of the case and we are prepared for the direct physical examination. We note the amount of the discharge, its color, its odor, its consistency, its constancy or its periodicity. We then undertake to determine whether there is a primary focus that is feeding the infection. We examine the nostrils for discharge, for the state of the mucous membrane and for obstruction. We transilluminate the sinuses. Comparatively often we find cloudiness on the same side as the purulent discharge. Oftentimes the sinuses are apparently clear. Do not let this stop you. No examination is complete without a careful x-ray of the sinuses by a thoroughly competent man. At the same time that the sinuses are x-rayed there should be a careful roentgenogram of both mastoid processes. This may show an area of necrosis either single or multiple. It may show the type of mastoid aptly termed by Mr. Arthur Cheatle the infantile type, with no cells in the mastoid.

For the sake of brevity I assume that where it is indicated the internist and the laboratory technician have been summoned to our aid. We are now prepared to launch the attack. If we find infected sinuses our first duty is to deal properly and efficiently with them. Many men have different ways of dealing with this problem, and I shall not here undertake to enter this controversial field further than to state that when it comes to a case of chronic infection in the sinuses I am more of a surgeon than I am a topical application man. I have had a long experience in the treatment of chronic sinus infection and on the whole it has been unsatisfactory. I have had a long experience in the surgical drainage of chronic sinus infection and on the whole it has been satisfactory. Faced with a chronic sinus infection complicated by a chronic discharging ear, I should not be inclined to spend a great deal of time trying to clear up the sinuses by treatment. Unless there was marked improvement of a character which gave promise of being permanent I should wish to proceed to surgical drainage. I have the feeling that it is a waste of time to continue to treat a chronic discharging ear in the presence of an unresolved sinus infection. My experience has been that after the sinuses are cleared up the chronic purulent infection of the middle ear will clear up spontaneously. Suppose we find that the patient has both an infected sinus and also a chronic mastoiditis? Here I should be inclined to spend

very little time in treatment. A chronic mastoid infection is like a chronic sinus infection. About the only thing that favorably influences a chronic mastoiditis is the eradication of broken down cells. Which should be attacked first, the sinus or the mastoid? Since I regard a mastoid as a greater menace to the life of the patient, it is my policy to clean out the mastoid first. I should not delay draining the sinuses any longer than it takes the patient to recover from the shock of the mastoid operation.

We have now outlined the treatment of chronic purulent otitis where it is a complication of infected sinuses and infected mastoid cells. What is our line of procedure where we find neither infected sinuses nor infected mastoid cells? In this case we are dealing with a condition that offers very little menace to life and health. Therefore we can afford to take our time and give the patient the full benefit of local treatment. Almost all these cases will respond to careful, persistent attention. These are the cases that have responded to zinc ionization. My feeling is that they will respond equally well to other forms of treatment.

During a long professional life I have seen many different types of treatment advocated. I still believe that in the presence of a chronic discharging ear which is not a complication of sinus or mastoid infection the first essential to healing is cleanliness and the most important aid to cleanliness is the universal solvent, water. I syringe the ear with a warm boric or saline solution and then spend a long time in drying it. After the ear is thoroughly dry I dust in either iodine powder or another antiseptic powder. I find in some cases that iodine powder is irritating. This type of treatment will cure many cases that we consider operative if the patient and the physician are equally faithful in the pursuit of the treatment. In my professional experience I have learned as much from patients who refused my advice as from those who have taken it. Sometimes the patient has suffered by refusing my advice and in other cases it is I who have had my eyes opened. I can recall five patients who had a chronic purulent otitis with unmistakable evidence of cholesteatomatous deposits associated with dangerous symptoms. I urged operation, but the patients refused. They all said they were willing to come to my office every day for treatment, but were not willing to be operated upon. They all came faithfully over a long period and received a cleansing treatment. The result was the same in all five cases. One day I would look in the ear and see a mass of black material which proved to be cholesteatoma. This was removed and there was more the next day. When the cholesteatomatous masses ceased to appear in the canal, the ear quickly dried up and in no case has there been any recurrence. I should not, of course, recommend this as routine treatment, because it is too time-consuming and uncertain, and there are not many patients who would be faithful enough to see it through. But if the patient refuses a radical operation it is well to know that these cases have been cured by treatment.

I think that aural polypi should never be attacked surgically. There is too much danger of lighting up an intracranial infection. For thirty-six years I have dealt with them by cauterization. The agent used is a saturated solution of potassium bichromate. After the ear is cleansed and dried thoroughly an applicator with a small cotton tip is dipped in the solution and applied to the polypus. A few applications will generally suffice to get rid of the polypi. This will aid drainage and promote healing.

In the foregoing discussion I have indicated my approach to the problem of chronic suppuration of the middle ear and mastoid. It is based on conclusions arrived at after sixteen years of investigation of middle ear and mastoid suppuration. It is with peculiar pleasure that I offer you these conclusions, as it was through this Section that I presented my first paper on this subject to the general profession, at the Miami meeting, November 19, 1929. My final conclusions are:

(1) At least 85 per cent of purulent infections of the middle ear and mastoid are the result of a purulent sinusitis, nearly always on the same side as the infected ear and mastoid.

(2) Ninety-one per cent of those having scarlet fever have a coincident infection in the sinuses. It is presumed from clinical symptoms that about the same per cent have a coincident sinusitis in epidemic influenza. The chances are that the other exanthematous diseases also have a high percentage of sinusitis.

(3) If 91 per cent of patients ill with scarlet fever and influenza have sinus disease and sinusitis also accompanies the other exanthematous diseases, then at one time or another practically the entire population suffers from sinus disease.

(4) A large percentage are left with a chronic sinus infection that tends to last throughout life unless diagnosed and treated.

(5) These chronic residual infections constitute a menace to the life, health and hearing of those so infected and make the victim a carrier, infecting others by contact and starting epidemics.

(6) What is diagnosed as an acute sinus infection is often an acute exacerbation of a chronic sinus infection.

(7) When the relationship is established between a chronic middle ear suppuration and a purulent sinusitis the first step in the treatment of the ear is the elimination of the sinus infection.

Case 1.—X-ray Case 8107. J. C., a boy, aged 16, had a discharging ear four months following a cold. He gave a history of abscess of the left ear following swimming. He consulted a specialist, who advised removal of tonsils and adenoids. The operation had no effect on the discharging ear. Mastoid operation was done. The mastoid did not heal until the left antrum was drained, after which it healed quickly. X-rays showed a densely cloudy left antrum and the cells of the left mastoid broken down.

Case 2.—X-ray Case 7090. In M. H., a boy, aged 16, both ears discharged for some time. Pain then developed in the left mastoid region and the left mastoid was operated upon. The maxillary sinuses were drained a year later, after which the chronic purulent discharge from the right ear ceased. X-rays showed that both maxillary sinuses were cloudy, and there was involvement of the left mastoid cells.

Case 3, Fig. 1.—X-ray Case 17994. E. T., a boy of 17, gave a history of diphtheria at three years of age. Since then he had a chronic purulent discharge from the right ear. There was a large perforation of the drum. He had a very bad color. X-ray by Dr. Shoulders showed a very cloudy right antrum, right ethmoid, right sphenoid and right frontal. He had a leaky heart.

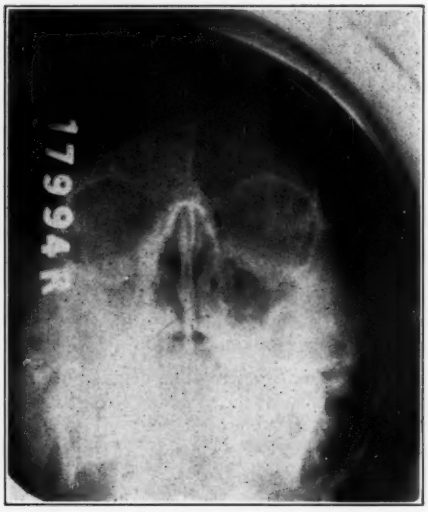

Fig. 1, Case 3

Case 4.—X-ray Case 527. Mrs. W. P. D., white, aged 50, was seen April 23, 1936. She gave a history of pneumonia at the age of 8, during which the left ear began to discharge. Pus ruptured through the mastoid bone without operation. The left ear had been discharging ever since. X-ray by Dr. Brown in 1933 showed a cloudy left antrum. Radical operation was done upon the left antrum at that time by Dr. Wesley Wilkerson. Following a recent attack of influenza the right ear had been discharging. The right antrum was cloudy under transillumination.

Case 5.—X-ray Case 18811. Mrs. H. T. A., white, aged 53, was first seen by my partner, the late Dr. George W. Hale, on March 3, 1890, for suppuration in the right ear, which began at the age of 2. The following year Dr. Hale did a mastoid on the right side. She had been treated for years for sinusitis by her deceased brother-in-law, who was an otologist. The scar of the old mastoid operation could be seen. The right antrum was cloudy under transillumination. Roentgenogram by Dr. Shoulders showed the right antrum, right ethmoid and right frontal very cloudy. Here is a case of sinusitis on which we have data running back fifty-one years, which is still in bad condition. This case shows the relationship between sinus disease and mastoiditis and it demonstrates forcefully the chronicity of sinus disease.

Case 6.—X-ray Case 19023-21351. Miss C. A., white, aged 33, was seen on April 22, 1935. She was the daughter of the previous patient. She had pneumonia at the age of 3, since which time she has had a chronic purulent discharge from the left ear. The discharge was intermittent. When she had a cold it lighted up and started discharging. The x-ray report on this case is negative, but when the ear is discharging pus can be seen issuing from the region of the natural opening of the antrums. It shows what we have all seen time and again, that not every case of sinusitis is demonstrable by the x-ray. This appears to be another case of the mother's being a carrier and infecting the daughter.

Case 7.—X-ray Case 18895-21131. H. L. M., white, aged 6, was seen May 6, 1935. He has had a discharging right ear since infancy. Roentgenogram showed the left mastoid cloudy, and both right and left antra very cloudy. But the saddest feature of this case is the roentgenogram of the lung showing that the child has bronchiectasis. Both antra were drained, following which discharge from the left ear ceased without treatment. Symptoms of bronchiectasis were ameliorated.

Case 8, Fig. 2.—X-ray Case 21038. T. C., aged 25, was first seen November 23, 1910. He had scarlet fever at 8 years of age, and since then had had a purulent discharge from both ears. A mastoid operation was done upon the left ear. The antrum was found to be filled with cholesteatoma. He refused operation upon the right ear. Under constant daily treatment, cholesteatoma came away in a mass and suppuration ceased. On October 7, 1935, he complained of arthritis in both arms and shoulders, which kept him awake at night. Roentgeno-

Fig. 2, Case 8

gram by Dr. Shoulders shows the right ethmoid and right antrum very cloudy. The left antrum is cloudy. This case demonstrates the relationship between sinusitis and middle ear infection and also shows a chronic sinusitis of more than forty years' duration still causing constitutional disease.

Case 9, Fig. 3.—Miss C. W., aged 54, consulted me Jan. 12, 1923. This is the most instructive case that I have ever had. The patient came on account of a profuse purulent discharge with very foul odor from both ears, which dated from an attack of influenza in 1918. She

Fig. 3, Case 9

was very deaf, and both antra were cloudy. Both antra were drained intranasally. I did not see her again until December, 1924, when she returned with an orbital abscess over the left eye. This was diagnosed empyema of the left frontal sinus with rupture through the floor into the orbit. The condition of the ears was unchanged. The orbital abscess was drained through an external incision. As soon as the acute symptoms subsided, I exenterated the left ethmoid labyrinth and made an opening into the frontal intranasally. The surgical nurse said it drained like an empyema of the chest. On January 25, 1925, I did a radical external operation on the left frontal. The floor of the frontal was practically destroyed by necrosis of the bone, and there was an exposure of the dura as large as a twenty-five-cent piece on the posterior wall. The patient's recovery was uneventful. I did not see her again for three months. When she came back the purulent discharge in both ears had cleared up completely. I saw her in July, 1934. There has never been the slightest return of the purulent discharge from the ears and her hearing has improved markedly.

March 2, 1937.—This patient has recently been in the office. When I saw her first in 1924 she had a profuse offensive purulent discharge from both ears, she could hear only very loud conversation. Both drums were destroyed. Following the drainage of both antra the exenteration of the left ethmoid labyrinth and an external operation on the left frontal sinus, the discharge from both ears ceased spontaneously. There has been no recurrence of the purulent discharge from either ear, she has had a complete reformation of both ear drums. Her hearing is greatly improved.

Case 10, Figs. 4 and 5.—X-ray Case 21804. S. H., aged 10, consulted me Aug. 6, 1936. She had scarlet fever at 8 years, with a profuse purulent discharge from the nostrils and ear during the attack. She had an extensive mastoid operation on the right ear. The right ear was still discharging a bloody purulent discharge. She had a mastoid sinus from which a small amount of pus exudes. Both maxillary sinuses were cloudy on transil-

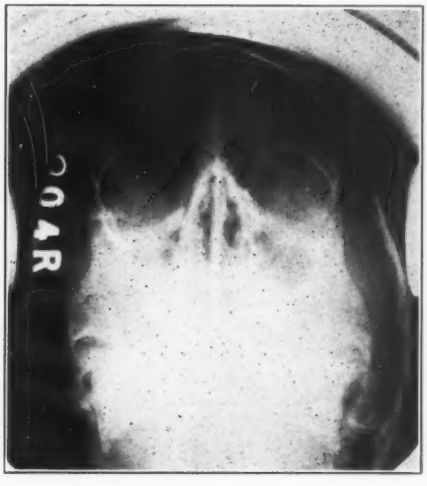

Fig. 4, Case 10

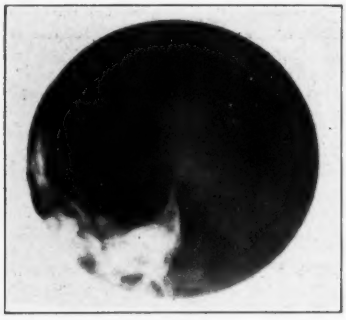

Fig. 5, Case 10

lumination. X-ray examination was made by Dr. H. S. Shoulders, who reported both maxillary sinuses cloudy, the right more than the left. I advised draining the maxillary sinuses, but operation was declined.

March 2, 1937.—This patient was treated three times weekly by packing the nostrils with antiseptic tampons. The post nasal space was treated with a solution of iodine in glycerine. After three months the sinus in the mastoid ceased. About a month later discharge from the ear ceased. The drum which was practically destroyed has completely reformed so that she has an entirely new drum. Her hearing is greatly improved. She has gained 15 pounds in weight. Her color has become normal. She has become quick and alert and full of vitality.

Case 11, Figs. 6 and 7.—X-ray Case 21925. H. E. K., aged 23, consulted me Sept. 22, 1936. He had had diphtheria in childhood, since which he had had a purulent discharge from the left ear. During the preceding few months drainage had been scanty and he had developed mental symptoms. He had had several convulsions, one very severe. Under transillumination both antra were cloudy. X-ray by Dr. Shoulders showed both antra

Fig. 7, Case 11

cloudy, the left very cloudy. The right mastoid was pneumatic and clear; the left mastoid sclerotic in type with all cells cloudy. The left mastoid was operated upon and both maxillary sinuses were drained.

Case 12, Figs. 8 and 9.—X-ray Case 21986. H. N. L. consulted me Oct. 13, 1936. He had a chronic purulent discharge from the right ear for a number of years. He had influenza while in the service in 1918. Afterwards the right ear began to discharge. The right ear canal was filled with profuse purulent discharge with a very bad odor. He had had a great deal of treatment without any effect on the discharge. He was treated by a specialist regularly for two years, but without effect. The specialist then dismissed him and told him not to waste any more time or money on the ear, as it was hopeless.

Both maxillary sinuses were cloudy on transillumination. X-ray by Dr. Shoulders showed the left frontal sinus large and cloudy. Both ethmoids were cloudy and both maxillary sinuses very cloudy. I advised drainage of both antra at once, to which he agreed. Both maxillary sinuses were drained at St. Thomas Hospital October 15. The right antrum, the one on the side of

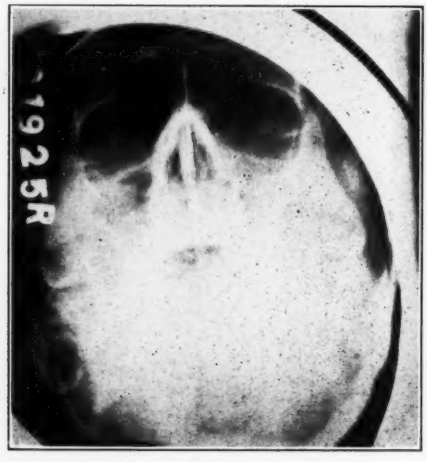

Fig. 6, Case 11

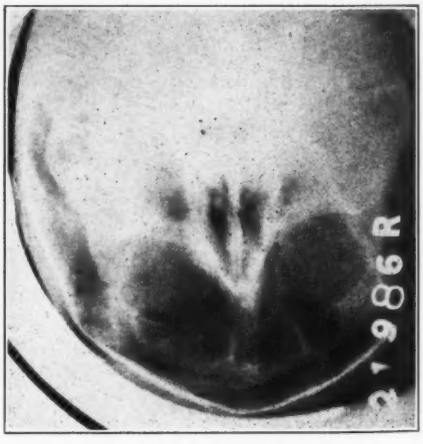

Fig. 8, Case 12

Fig. 9, Case 12

the discharging ear, contained a great deal of free pus. The mucous membrane of the left maxillary sinus was polypoid. He left the hospital on October 17 and came to the office October 19. There was a little pus in the right ear, which was cleansed, and an antiseptic powder insufflated. He came again on Tuesday. The right ear was entirely dry, a condition that had not existed in years. Since then the ear has remained continuously dry.

March 2, 1937.—The ear has remained continuously dry. The ear drum which was almost completely destroyed has entirely reformed, so that he has an entirely new ear drum, like *Case 10*. His hearing has greatly improved. He has recovered from his rheumatism, from which he was badly crippled, both knees being swollen and very painful. He is now pursuing his business with normal activity.

DISCUSSION (*Abstract*)

Dr. E. W. Rucker, Jr., Birmingham, Ala.—Some years ago Dr. Cullom called to our attention the frequent occurrence of infection of the nasal accessory sinuses concurrent with infection of the middle ear and mastoid, and when the nasal infection cleared up the ear cleared up also. This observation appealed to me, and I began to look for the nasal infection in these cases, and the more I looked for them the more I found, and the more attention I paid to the sinuses the better results I got in the ear. This should not seem strange, for in reality the middle ear is only another nasal accessory sinus, and what would affect one should affect the other.

I cannot emphasize too strongly Dr. Cullom's plea to you to go into the history of the case thoroughly and the study of the general condition of the patient. Particularly the last should be gone into from the biochemical or autonomic nervous system standpoint. Those of you who had the privilege of hearing Dr. Blackmar's paper last year on the N. C. G. level and are not taking advantage of this line of attack are overlooking a tremendous aid in therapy.

As regards the local treatment of these chronic ear infections, I believe if we spend more time in cleaning every vestige of debris, of whatever nature, from the ear, it makes little difference what form of medication we use. I have gotten into trouble from the use of dyes that leave a precipitate in the ear, therefore I have discontinued their use, as they interfere with drainage, which is essential. I prefer a dry cleaning, either with cotton or suction, to the irrigation, but there are cases that you will have to irrigate, especially those of attic involvement with cholesteatoma.

One of the most difficult types to handle is that in which a fungus infection is present. Dr. Searcy, of Tuscaloosa, in conjunction with the laboratory of the University of Alabama, has done a great deal of experimental work with all kinds of drugs and finds that thymol is the best fungicide in these cases. He advocates 1 per cent thymol added to boric acid and iodine powder for office use, and 1 per cent thymol added to drops for home use.

Without question there are cases of the so-called dangerous type that require a radical mastoid without delay, but I am glad to see the trend is more to the conservative side, and the indications for the radical operation are not what they were.

I cannot refrain from calling your attention to the use of a drug, insulin, in small doses, two to three units two or three times a week. This is especially valuable if there is any tendency to polyp formation, or a "water logged" look to the mucous membrane. The only drawback to using insulin is that the family physician will accuse you, as he has me, of practicing general medicine.

Dr. John R. Hume, New Orleans, La.—Despite a wealth of accumulated knowledge concerning the chronic suppurative ear and mastoid, it must be recognized by workers in our field that its clinical application has been sorely neglected. Patients stream into our offices stating in their histories that they have been advised to "keep the ear clean and let it run."

The pathologic condition existing in these cases presents a serious menace to health, to function and to life itself. As our essayist says, chronic aural suppuration is secondary to an infection in the nose, sinuses or pharynx. The focus should be discovered if present; in the tubo-tympanic type it must be determined if we may expect a cure. In those cases the discharge is mucopurulent in type and the perforation will be found anterior or meso-tympanic. The discharge, at times scant, becomes profuse with acute exacerbations with each chronic process in the nose or paranasal spaces. This type of case will frequently be cured by removal of the primary focus. Zinc ionization is effective in this type of case only. I prefer, however, after cleansing the external canal and tympanum carefully to fill the canal with silver nitrate, 3 per cent, or zinc sulphate, 2 per cent, and gently force it into the pharynx by way of the eustachian tube with pressure from a fistula bag. After three or four treatments on alternate days the ear is often found to be dry. Radical aural surgery is not indicated in these cases and serious intracranial complications are rarely noted.

If we find the perforation located in the posterior-superior margin indicating involvement in the region of the antrum or in the flaccid membrane, the problem becomes a more serious one. Ofttimes a search of the sinuses, nasopharynx and tonsils fails to reveal the focus. Time has removed the primary disease, but left a very definite and sometimes destructive lesion in the tympanic cavity and surrounding structures.

Before any treatment is instituted the location and extent of the involvement must be determined. Routinely we should determine the location and sign of the

perforation; the character of the discharge, as evidencing cholesteatomata or bony necrosis. The presence of polypi and granulations may be noted. Extension of the process into surrounding structures should be determined. The fistula and caloric tests will determine possible labyrinthine involvement, a positive fistula test suggesting a circumscribed lesion; a failure of reaction to the caloric test suggesting a latent lesion. The hearing impairment should be measured. X-ray studies of the mastoid and petrous regions should be made.

A failure to neglect any of these procedures may result in our failure to suggest proper treatment.

With the information such a routine examination will furnish we will know which case should be treated conservatively, which case may be treated conservatively, and which case must be at once submitted to radical surgery.

Dr. Cullom (closing).—Dr. Rucker's references to the use of thymol and insulin are new to me and very interesting.

If x-rays are made of sinuses of patients upon whom one is planning to do a radical mastoid, one will find a surprising number with a purulent sinusitis on the same side. Surgical drainage of these infected nasal sinuses will obviate the necessity of a radical mastoid in a fair number.

Here are three cases of chronic purulent otitis media that had resisted all forms of treatment. It was found that all three were suffering with chronic purulent sinusitis. When the chronic purulent sinusitis was relieved by surgical drainage in two cases and by treatment in the other, the chronic purulent discharge from the ears ceased spontaneously. The cessation of the discharge from the ears was followed by complete reformation of the ear drums in all three cases.

MYCOSIS FUNGOIDES*

By JAMES K. HOWLES, M.D.
and
JOHN CONNELL, M.D.
New Orleans, Louisiana

Since Alibert, in 1812, first described the clinical entity known today as mycosis fungoides, there has been considerable confusion associated with its variable clinical manifestations. Advances in histopathological studies have helped to create some degree of order from the chaos of uncertainty and ignorance enshrouding this disease. So many theories exist as to the etiologic factors of this disease entity that a review of the literature only increases the magnitude of the enigma.

*Read in Section on Dermatology and Syphilology, Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

*From the Department of Dermatology and Syphilology and Department of Pathology, Louisiana State University Medical Center and State Charity Hospital, New Orleans.

Fraser[1] lists nine different views which have been advanced to explain its nature. First and foremost is that it is a granuloma. The other views include cutaneous lymphadenoma, lymphosarcoma and lymphemic lymphomatosis and even sarcoma superimposed on some chronic dermatosis. Symmers[2] has sponsored the theory that mycosis fungoides is a cutaneous manifestation of some systemic disease. He believes that the condition is the cutaneous expression of at least three different diseases of the lymphatic system. Hodgkin's disease, a variety of round cell sarcoma arising from the connective tissue reticulum of lymph nodes or elsewhere, and lymphosarcoma.

Since Virchow's original description of leukemia, the correct classification of the various manifestations of the lymphatic system has been a problem of major importance to medical investigators. There has been a concerted effort in recent years on the part of both clinicians and pathologists to group under one general head all of the cutaneous diseases of lymphatic origin. Keim,[3] in 1924, suggested that the multiple clinical pictures associated with true lymphadenoses should be regarded as the variable cutaneous expressions of the lymphoblastomas. Internists and hematologists were quick to see the value of such a classification and there has been an increasing interest in the problems associated with the various clinical manifestations of the diseases of the hemopoietic system and a growing tendency to regard these various clinical pictures as part of the same disease process. The true lymphadenitic infiltration of the skin occurs most commonly in association with the forms of lymphatic hyperplasia, generally referred to as granuloma fungoides, Hodgkin's disease, leukemia and lymphosarcoma. In addition to these certain rare transitional forms or subvarieties of these are included. There still exists a great deal of diversity of opinion in the classification of different cases. This difference of opinion is probably due to the variation in the cutaneous manifestations of this group and the possibility of one variety's merging into another. Cases appear from time to time that possess features of two or more of these diseases. This classification has established a uniform terminology, if nothing more, and while there still exists considerable variance of opinion as to the exact nature of these lymphoblastomas, the adoption of such a grouping has established the certainty of the interrelationship of these diseases.

It seems the next step is definitely to establish the true etiologic nature of the components of

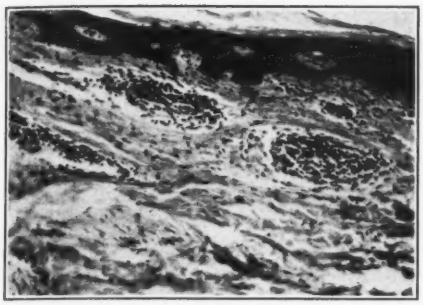

Fig. 1

This earliest lesion has epithelium showing moderate hyperkeratosis and some parakeratosis. The basilar outline is distinct. The upper layers of the cutis are moderately involved, with an apparent inflammatory reaction. Deeper and perivascularly, there is a more marked reaction.

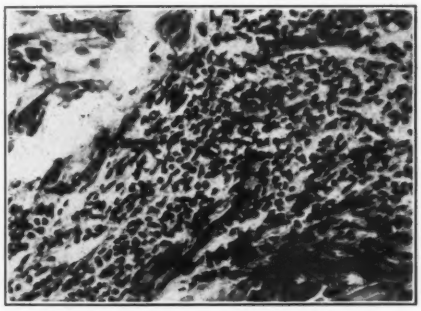

Fig. 2

A high power of an inflammatory focus of Fig. 1. These inflammatory cells are, in the main, round, homogeneous, and small. Typical non-specific inflammatory cells.

the lymphoblastomatous group. I concur with Montgomery[4] that until more is known regarding the etiology of the different diseases concerned, the attempt should still be made to classify them as separate but related entities.

The appearance of granulomatous lesions in the skin during the course of those diseases had led Unna,[5] Ormsby,[6] Macleod[7] and others to believe that the probable etiologic agent is infectious. Supporting this view is the constant finding in the histologic sections of these lesions of connective tissue proliferation with an accompanying infiltrate of lymphoid cells, suggesting chronic infectious granuloma. The extensive bacteriologic investigation carried on by Fox[21] and Frasier to substantiate the infectious etiology has failed to establish any definite etiologic organism.

There is a growing enthusiasm for the belief that all of these lymphoblastomas are genetically

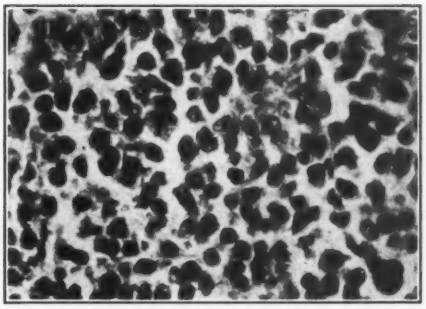

Fig. 3

Section removed from subcutaneous tumor nodule at time of autopsy shows the true neoplastic nature of the final growth, manifested by cells having large sized nuclei, which are hyperchromatic and are undergoing mitotic division.

related neoplasms involving the lymphatic tissues. Montgomery[4] considers mycosis fungoides to be a neoplasm originating in the reticulo-endothelioma of the skin. Warthin's[9] extensive histological studies caused him to classify them all as related neoplasms. This seems very probable, as they all show metastasis and infiltrative tendencies. In fact, the same histologic picture that is interpreted by the believers in the infectious origin as inflammatory, namely, connective tissue proliferation and infiltration of lymphoid cells, as pointed out by Wile,[8] has been considered by an increasing number of histopathologists as evidence of neoplastic activity.

Keim[10] and Fraser[11] point out that even in the early inflammatory lesions small groups of neoplastic cells may be found after extensive examination of the sections. Keim's[10] clinical viewpoint concurs with his colleague Warthin's pathologic opinion, namely, that these diseases are all related and have in common a cell of the lymphocytic series as a type cell and that histologically and clinically the variation in the lymphoblastomas is closely allied to the degree of differentiation of these cells. Probably this accounts for the mutations or interchanging of clinical types of lymphoblastomas.

It is not the purpose of the essayist to endeavor to establish the viewpoint of either group as to the etiologic nature of the lymphoblastomas. The literature and various points of view regarding these diseases are covered by Herxheimer and Martin,[12] Gaus,[13] Wile,[14] Keim,[15] Wise,[16] McCarthy,[17] Cole,[18] Symmers,[19] Schoenhof,[20] and others.

Mycosis fungoides is still an uncommon cutaneous disorder and on account of its variable clinical and histologic features it has presented

most perplexing problems. Because of the bizarre features of this case it seems to merit presentation, for as Lane and Greenwood[23] have pointed out that while the unusual cases in this group may complicate the picture at present, they may contribute with the summation of evidence to the clarification of the relationship with the group.

Ormsby,[6] Unna,[5] Macleod,[7] Montgomery[4] and others refer to the relative infrequency with which the internal organs, in mycosis fungoides, of the body are involved by the lymphoblastomatous process. It is because of the unusual complications which this case presented that this report is submitted.

REPORT OF CASE

H. K., a white male, aged 54, a native of Alabama, married, with no children, gave an unimportant past history, having had measles, scarlet fever and chicken pox as a child, but no previous serious illness. He first noticed in 1929 patches of scaly dermatitis which involved the arms, lower abdominal wall and calves of the legs. The lesions itched intensely. They had no seasonal predilection. The lesions had been diagnosed ringworm and eczema and various other conditions by different physicians. They disappeared under topical therapy and superficial x-ray treatment and left no scars except a slight hyperpigmentation of the skin after resolution.

When first seen by me in the year of 1932, the patient had several circumscribed lesions of the calves of both legs, thighs and the lower abdominal wall. The lesions were hyperpigmented plaques raised only slightly above the skin surface. The color and circumscription of the lesions suggested either a morphea or maculoanesthetic leprosy. There was a slight amount of anesthesia present. Scrapings were made of the lesions, but examinations for Hansen's bacilli were repeatedly negative. A bland ointment was prescribed, as the patient complained of only slight itching.

The patient returned to the skin clinic several weeks later, July, 1933, and the plaques previously described were more elevated, had an inflammatory base and were covered with fine scales. Itching was intense. Even in the face of the unusual distribution and the pruritis a diagnosis of psoriasis was ventured. A biopsy was taken and the histological picture was entirely different from the classical one of psoriasis. Only a chronic inflammatory reaction could be detected histologically. There were no lesions of the scalp or other sites of predilection characteristic of psoriasis. Parapsoriasis was considered. Scrapings of the lesions and examination of scales failed to reveal any fungi on microscopic examination. The blood Wassermann and urinalysis were negative. The patient was treated with superficial x-ray and keratolytic ointments locally and potassium arsenatii by mouth. The lesions subsided within a month. Four months later (December 27, 1933) the patient was admitted to the hospital with the following history:

Twelve days before, he had developed a stye on the lower lid of his left eye. The following day the stye was more inflamed, so he went to a local clinic and was advised to apply hot compresses to the involved area. He did this at frequent intervals and believed that he applied the compresses too hot, as his left eyelids became greatly inflamed and began to swell until within a few days they were as large as a lemon. The patient was advised to go into another hospital for x-ray treatments. On admission to the hospital the lesion of the left eye resembled the massive traumatic lesion which follows a severe blow. There were marked edema, lymphostasis and ecchymosis of the entire periorbital supra and infraorbital area. The picture was not unlike one of fulminating anthrax. He had lost the sight of the affected eye. The intraorbital pressure was intense.

An x-ray picture of the head and face at that time showed marked haziness and indistinctness of the left orbit, but no actual evidence of destruction in the orbital wall. A view of the chest showed calcified nodes in the peri-hilar region, but no sign of vesicular involvement of the lung. A biopsy from the left eyelid following the superficial x-ray treatments revealed a rather unusual structure which made differentiation between round cell sarcoma and a malignant tumor arising from the retina very difficult.

Because of the previous unusual cutaneous lesions

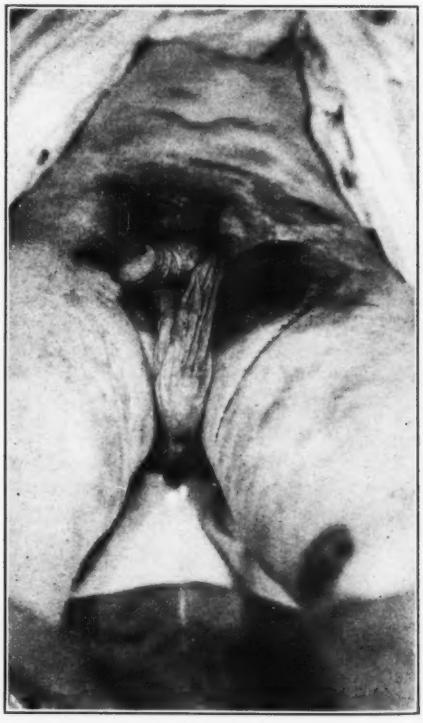

Fig. 4

Photograph shows nodular lesions of thighs and abdomen. Some of these lesions were fungating and presented the typical, clinical picture of the mycotic stage of mycosis fungoides.

which the patient had manifested, lymphoblastoma was considered at that time, but the lesion of the eye suggested nothing of that nature to the examiner. The patient complained of severe pains of the left eye and of the head. He was given superficial x-ray treatment to the left eye and almost immediate relief from his pain was noted.

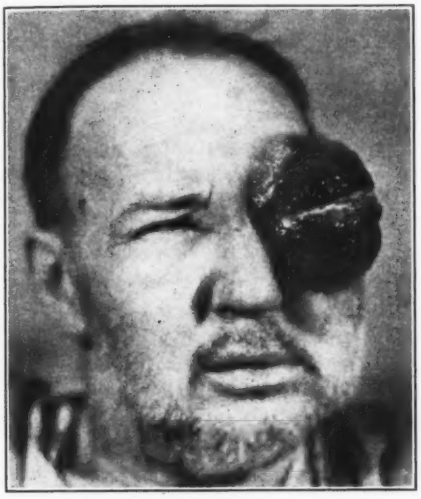

Fig. 5

Fig. 6

Figs. 5 and 6 show the early lesion of the left eye described in this article. The massiveness of the lesion can be seen. This area of inflammation subsided under small doses of superficial roentgen therapy.

The cutaneous lesions had now reappeared at sites where they were previously noted on the skin five months before. There were several circumscribed, raised plaques very much inflamed, involving the lower abdomen, the inguinal region and the anterior cervical areas. The lesions were covered by a fine silvery scale and itched intensely. They looked not unlike psoriasis. There was no involvement of the scalp. No enlargement of the lymph nodes were detected. The mucous membranes were not involved. The size of the plaques varied from 7 to 18 cm. in size. A biopsy taken of one of these lesions (February 2, 1934) in the groin showed the typical histological picture of lymphosarcoma, although the clinical picture was compatible with psoriasis.

Except for the cutaneous lesions noted and the eye lesions, the physical examination of all systems was entirely negative.

The laboratory findings on admission shall be listed in the resume to be given shortly.

COURSE OF THE DISEASE AND TREATMENT

Beginning January 2, 1934, and continuing until January 16, 1934, a course of five treatments both of filtered and unfiltered x-ray was given to the area around the left eye. Six hundred and twenty-eight R units of unfiltered and 872 R units of filtered x-ray were given to that area. The lesion of the eye subsided considerably after the x-ray treatments were begun. The relief from the excruciating pain was very striking.

On January 20, 1934, the patient complained of severe pains in both legs. X-ray of the vertebrae and long bones showed nothing abnormal. The patient continued to complain of the pain in the head and legs, however.

The cutaneous plaques in the groin became nodular until they were the size of a hen's egg and began to ulcerate. Superficial x-ray therapy caused some resolution of these nodules. The patient complained of less itching of the involved areas. His general condition was growing worse. He became hoarse and the hoarseness increased gradually until by the last of March, 1934, there was definite evidence of laryngeal obstruction. The patient became dyspneic and very cyanosed.

X-ray of the chest taken at this time showed the same fibrosis and calcification of the left hilar region and the left apex as were seen in the x-ray plate two months previously. It became necessary to tracheotomize the patient March 28, 1934, because of the dyspnea and laryngeal obstruction. On April 19, 1934, after the dyspnea had been relieved by the tracheotomy a laryngoscopic examination was performed and a biopsy of the tumor mass in the larynx was made. A histologic diagnosis was made of a very malignant growth of the type of carcinosarcoma. Numerous yeast-like bodies were seen in the growth.

Because of the increasing weakness several transfusions of whole and citrated blood were given, but to no avail. The patient developed rales throughout the chest, associated with marked dyspnea. He expired May 19, 1934.

A resume of the laboratory examinations done while in the hospital is as follows: repeated urinalyses were negative, except near the time of exodus, when a few granular casts were seen.

BLOOD COUNTS	12/29/33	2/6/34	2/18/34	3/12/34	4/10/34
Total R. B. C.	3,970,000	4,750,000	3,600,000	3,250,000	3,000,000
Total W. B. C.	6,600	10,200	12,500	9,500	10,000
Hb. per cent	70	75	65	65	60
S. M.	23	17	26	24	20
L. M.		4	6	5	6
N.	77	76	68	70	73
Eosinophils per cent		3		1	1
Pathologic cells	none	myelocytes			

Blood Chemistry	12/30/33	2/8/34
N. P. N.	30 mgm. 100 c. c. blood	31 mgm. 100 c. c.
Urea nitrogen		11.2
Blood sugar	100	100

Blood culture January 9, 1934, negative.

NECROPSY

The body is that of a fairly well developed, markedly emaciated male, 54 years of age. The skin in many areas, particularly the cervical and inguinal regions, shows depigmented scars. On the anterior abdominal wall, a subcutaneous tumefaction is seen which is raised approximately 0.5 c. c. above the surface and measures approximately 2 cm. in diameter. This tumor is reddish white in color and quite friable. No other subcutaneous nodules are present.

Besides the marked parenchymatous changes in all the visceral organs, there are only two important pathological alterations.

The larynx shows an infiltration of the left wall involving and destroying the vocal cords. This infiltration caused a bulging mass to protrude into the pharynx, which became the site of a superficial ulceration. The lungs have nodules diffusely distributed over the surfaces of all lobes, the larger nodules measuring 4 cm. in diameter. These are deep red in color, vascular, and very friable. The mediastinal lymph nodes are moderately enlarged, but do not appear to be of a malignant nature.

HISTOPATHOLOGY

From the histopathological aspects of the case, we see an unusual transition, not identical with that usually described as typical of mycosis fungoides. The early involvement of the lower layers of the epithelium is conspicuously lacking and the so-called inflammatory activity is confined not only to the upper layers of the cutis but perivascularly deeper in the cuticular layer.

The first biopsy, which was taken at the time of the psoriasiform lesions, as previously described, was taken approximately seven months prior to the death of the individual. This biopsy showed an epidermis revealing little or no hyperkeratosis, quite marked parakeratosis and quite active proliferation of the basal layer. This, however, in no way appeared specific. The cuticular layer, on the other hand, showed an infiltration throughout the cutis more prominent in the upper layers and perivascularly. The infiltration was polymorphous in character, consisting in the main of small lymphocytic-like cells, apparently mature. There were a few scattered plasma cells, epithelial cells, fibroblasts and a very occasional eosinophil. The interpretation at this time was one of non-specificity.

Four months later a second biopsy was taken in another region of the skin, at which time the microscopic picture had changed but little. We saw occasional large mononuclear cells in our inflammatory process and the suggestion of a clumping of the lymphocytic-like cells.

The third section was recovered two weeks later from the lesion described that occurred in the eye. This lesion, for the first time, appeared to have taken on some malignant characteristics and is probably classified in the so-called intermediate or infiltrative stage of the disease. In these sections we see what we interpret as malignant cells which are small, round, show hyperchromatic nuclei and a very small amount of cytoplasm.

A month later the fourth section was removed from the first actual tumor mass that appeared on the skin. This section showed, for the first time, invasion of the epidermis. The basal layer is destroyed in many places. There is an infiltration of foreign cells throughout the lower layers of the dermis. These cells almost completely replace the upper portions of the cutis, likewise involving somewhat the lower portions. The invading cells give a rather regular picture of cells too large for inflammatory, yet smaller than the ordinary malignant cells. They are, for the most part, round, have large irregular nuclei, showing hyperchromatism; here and there can be seen pseudo giant cells containing three or four nuclei. At this stage we believe we are definitely dealing with a tumor showing all the malignant characteristics of invasion and destruction.

Three months later, a section was removed from a laryngeal tumor which showed definite infiltration and destruction of the normal architecture with malignant looking cells. These were in all characteristics similar to those described in the last biopsy.

The sections removed at the time of autopsy of the individual from the skin nodule on the abdominal wall and from metastatic lesions within the lung showed them to be identical. The lung was invaded in nodular formation without, however, the complete destruction of the alveolar walls which is usually seen in the advancement of malignant growths. This tumor seemed rather to infiltrate and widen the interalveolar spaces and, in some few instances, to break through them and fill the alveolar spaces. The tumor itself, at this stage,

shows a definite change in its characteristics. The cells have become larger, resembling those of a large round cell sarcoma. The stroma is a uniform, extremely fine reticular network instead of the coarser one afforded by the previously invaded organs. The cells in general are round, but many are oval in shape. The nuclei are enormous in size and constitute almost the entire cell. Within the nuclei can be seen one or two nucleoli. The chromatin stains extremely deep and is grouped in clumps, and mitotic activity is plainly evident. The sections from the skin showed a very similar picture with almost complete destruction of the lower layers of the epidermis and complete replacement of the cuticular layers with this tumorous tissue. Occasional plasma cells were seen in these tumors, but no eosinophils were seen throughout the sections.

In resumé: an initial lesion was interpreted as an inflammatory one involving the cutis which progressed and later involved basilar portions of the epidermis, still it appeared more inflammatory than neoplastic in nature. Then: a stage occurred in which, with the inflammatory cells, there appeared larger round cells with many beginning malignant characteristics. This lesion in turn progressed to one of frank neoplasia with all the characteristics suggestive of such a growth.

COMMENT

This case confirms the common opinion, that of the diseases primarily affecting the skin, none can appear in so many different forms or is misdiagnosed so often as mycosis fungoides.

Dermatological literature is replete with reports of cases which began with lesions simulating psoriasis clinically, but few have confirmed this clinical impression with histologic examinations. Fox,[24] in 1913, reported a case which began as psoriasis and changed in 25 years into a typical case of mycosis fungoides.

Stowers[25] reported a case in which the clinical picture had varied so much that it had been mistaken for eczema, leprosy, lupus and psoriasis. Allgeyer,[26] Wolters,[28] and Whitfield,[27] all describe cases of mycosis fungoides which early were mistaken for psoriasis. Volckers[29] reported a case which clinically resembled both psoriasis and syphilis. Mannino,[30] as early as 1880, reported a case in which a diffuse psoriasis appeared during the course of a mycosis fungoides eruption (tumor stage).

The case herein presented resembled psoriasis clinically in certain respects, but the classical features of a psoriatic eruption were missing and the histopathologic features were not compatible with psoriasis.

Regarding the acute flare-up of the skin lesions following the appearance of the inoffensive looking lesion on the left eye, which was diagnosed as a stye by a competent ophthalmologist, one is reminded of the reports of mycosis fungoides, which have developed following injury. Eller and Rein[31] report a case following an insect bite. The authors mention the possibility that the insect bite may have been coincidental with the development of the primary mycosis fungoides tumor. Probably these original patches of mycosis fungoides, which are so often misdiagnosed, are the primary lesions of the disease, which Hallopean[32] described as "the chancre of this disease" and which Eller and Rein[31] mentioned in their excellent article on this subject. The same authors quote Audrey[33] as concluding that in mycosis fungoides with a solitary tumor appearing on healthy skin, the tumor represented the chancre stage of the disease, and that one could expect a cure if the primary lesion was excised early enough.

The entire clinical picture in this case changed following the appearance of the eye lesion and there was a complete transition in the histologic structure also, as no longer did the inflammatory phase predominate, but a neoplastic cycle seemed to be developing which changed rapidly, and soon presented a typical malignant picture.

Still another feature of note was the absence of any evidence in the blood picture which would have created a suspicion of the true nature of the early skin lesion. Ketran and Goodman[35] quote Bazin[36] as early as 1862 as describing the most common clinical signs as only cutaneous lesions without visceral manifestations or alterations in the blood picture and in most instances presenting no evident involvement of the lymphatic system. Lane and Greenwood[34] report 23.4 per cent of their cases showed abnormal blood pictures. A perusal of the literature shows, however, that the condition may occur with or without changes in the circulating blood stream. In the majority of cases abnormalities are absent at the onset and develop later in the course of the disease. A marked lymphocytic deviation sometimes occurs only shortly before death.

No lymphadenitis was noted in our case until after the tumefaction occurred on the skin. Probably an early biopsy of the glands may have shown some changes which would have given us a lead during the questionable stage. A histologic study of the glands at autopsy revealed the hyperplastic changes comparable to what is generally reported.

The involvement of the larynx and lungs in this case is atypical. Although Minot and Isaacs,[37] in reporting on 477 cases of lymphoblastoma, emphasized the importance of abdomi-

nal symptoms occurring as an initial complaint or early in the disease, these authors include in their classification of lymphoblastoma cases of lymphatic leukemia, its aleukemic form (pseudoleukemia), lymphoblastoma and Hodgkin's disease. No mention is made of abdominal or other visceral complaints in mycosis fungoides.

The consensus of opinion seems to be that mycosis fungoides is a cutaneous disease and the visceral complications are extremely rare. Probably as a result of more study it will be found that associated with the mutation of this malady an increase in systemic involvement will be observed.

The histopathologic changes are self-evident and no effort shall be made to establish either the infectious or neoplastic theories of origin.

Certainly, however, we do not concur with Frieboes,[38] who considered eosinophils to be characteristic of all stages of the disease.

It seems the only feature of this paper which justified its presentation, if such justification is due, is the histopathologic studies which enable us to observe the tissue changes concurring with the clinical picture. This certainly emphasizes the importance of early and frequent biopsies and coordination of clinical and histopathological findings.

SUMMARY

(1) A case of mycosis fungoides is presented showing the clinical and histopathologic findings from onset of the benign lesions to autopsy.

(2) Atypical visceral complications were observed and necessary laboratory tests recorded.

(3) The mutations are significant because of the combined clinical and pathologic studies.

BIBLIOGRAPHY

1. Fraser, J. F.: Jour. Cutaneous Dis., **35**:793, 1917.
2. Symmers, Douglas: Arch. Dermat. & Syph., **25**:3 (Jan.) 1932.
3. Keim, H. L.: Arch. Dermat. & Syph., **10**:576 (Nov.) 1924.
4. Montgomery, H.: Arch. Derm. & Syph., **27**:253, 1933.
5. Unna, P. G.: The Histopathology of the Disease of the Skin, p. 509. New York: Macmillan Company, 1896.
6. Ormsby, O. S.: and Finnerud, C. W.: Mycosis Fungoides. Arch. Dermat. & Syph., **27**:631 (April) 1933.
7. Macleod, J. M. H.: Diseases of the Skin. London: H. K. Lewis, 1933.
8. Wile, U. J.; and Stiles, Frank, Jr.: J.A.M.A., **104**:532 (Feb.) 1935.
9. Warthin, A. S.: Ann. Surg., **93**:153 (Jan.) 1931.
10. Keim, H. L.: Arch. Dermat. & Syph., **19**:533 (April) 1929.
11. Fraser, J. F.: Arch. Dermat. & Syph., **11**:425 (April) 1925.
12. Herxheimer, K.; and Martin, H.: Mycosis Fungoides in Jadassohn, J.: Handbuch der Haut und Geschlechtaskrankheiten. Berlin: Julius Springer, **8**:174, 1929.
13. Gans, Oscar: Histologie der Hautkrankheiten, **1**:557. Berlin: Julius Springer, 1925.
    The Skin Picture in Hematopoietic Diseases. Arch. Dermat. & Syph., **16**:1 (July) 1927.
14. Wile, U. J.: Cutaneous Manifestations of Systemic Disease. Ann. Int. Med., **5**:1103 (March) 1932.
15. Keim, H. L.: Universal Leukemia Cutis. Arch. Dermat. & Syph., **10**:579 (Nov.) 1924.
    The Lymphoblastomas: Their Interrelationships. Ibid, **19**: 533 (April) 1929.
16. Wise, Fred: Lymphadenosis Cutis Universalis, Associated with Generalized Erythrodermia and Atrophy of the Skin. Jour. Cutan. Dis., **35**:668 (Oct.) 1917.
17. McCarthy, Lee: Histopathology of Skin Diseases, pp. 283 and 483. St. Louis: C. V. Mosby Co., 1931.
18. Cole, H. N.: The Cutaneous Manifestations of Hodgkin's Disease: Lymphogranulomatous. J.A.M.A., **69**:311 (Aug. 4) 1917.
19. Symmers, Douglas: The Association of Certain Cutaneous Lesions with Diseases of the Hemopoietic System. Jour. Cutan. Dis., **37**:1 (Jan.) 1919.
20. Schoenhof, Siegmond: Die Lymphogranulomatose der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, **8**:271. Berlin: Julius Springer, 1929.
21. Fox, Herbert: Amer. Jour. Path., **3**:544 (Sept.) 1927.
22. Fraser, J. F.: Jour. Cutan. Dis., **35**:793 (Dec.) 1917.
23. Lane, C. G.; and Greenwood, A. M.: Arch. Derm. & Syph., **27**:643 (April) 1933.
24. Fox, Howard: J.A.M.A., **61**:5-330 (Aug.) 1903.
25. Stowers, J. H.: Brit. Jour. Dermat., **15**:47, 1903.
26. Allgeyer, V.: Ach. p. le sc. med., **25**:235, 1901.
27. Whitfield, A.: Brit. Jour. Dermat., **10**:153, 1898.
28. Wolters, M.: Stuttgart, 1899.
29. Volckers, A.: Ueber Granuloma Fungoides der Haut. Inaug. Dissert., Munchen, 1893.
30. Mannino, L.: Gior. Ital. d. mal. ven., **17**:348, 1882.
31. Eller, J. J.; and Rein, C. R.: Arch. Dermat. & Syph., **27**: 807 (May) 1933.
32. Hallopean, H.: Traite pratique de dermatologie. Paris: J. B. Balliere et fils, 1900.
33. Audrey, M. C.: Bull. Soc. franc. de dermat. et. syph., **35**:432 (May) 1928.
34. Lane, C. Guy; and Greenwood, Arthur M.: Arch. Dermat. & Syph., **27**:651 (April) 1933.
35. Ketran, Lloyd W.; and Goodman, M. H.: Arch. Dermat. & Syph., **24**:759 (Nov.) 1931.
36. Bazin, A. P. E.: Lecons theoriques et cliniques sur les affections cutaneous artificielles, p. 372. Paris, 1862.
37. Minot, G. R.; and Isaacs, Raphael: Amer. Jour. Med. Sci. (Aug.) 1926.
38. Frieboes, W.: Grandriss der Histopathologie der Haulkrankheiten, **2**:222. Leipsiz: F. C. W. Vogel, 1924.

DISCUSSION (*Abstract*)

Dr. Winston U. Rutledge, Louisville, Ky.—First, there is the importance of taking biopsies. This is especially necessary in all chronic skin conditions which resist the ordinary therapeutic measures and which are prone to recur, for as Dr. Howles has stated, mycosis fungoides may in its early stages simulate psoriasis, parapsoriasis, ringworm, syphilis, eczema and even leprosy. Because of this clinical mimicry, biopsy study may be the only means available for arriving at a correct diagnosis. Again, where a single biopsy gives inconclusive information, additional biopsies made simultaneously from different lesions, or subsequent ones taken later during the progress of the disease, may be of much diagnostic aid. Where it is possible, biopsy studies should be made also from any associated enlarged lymph nodes.

I think that Dr. Howles is wise in not entering into the controversy which exists at present regarding the etiology of this disease and its classification in the group of so-called lymphoblastomas. Much progress has been made in recent years along these lines, but a better understanding of cellular physiology and pathology will have to be achieved before such questions can be settled with any degree of finality.

I feel that the acute fulminating lesions developing in the patient's left orbit, the unusual involvement of the larynx and pulmonary tissue, and the absence of any outstanding changes in the cellular elements of the blood more than justify the presentation of this instructive case report.

THE NON-OPERATIVE TREATMENT OF FRACTURES AND DISLOCATIONS OF THE SPINE*

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During the major part of my experience as a fracture surgeon, the diagnosis and treatment of frank fractures and dislocations of the spine has been quite unsatisfactory. Until recent years the entire problem was approached with apprehension. The resultant morbidity and mortality in most cases that were subjected to open operation fully justified such a feeling.

If patients with fractured spines and dislocated vertebrae did not die from shock in the first few hours, or from high cord paralysis a little later, then cystitis, pyelitis, sepsis, exhaustion and death, in the order named, usually followed in the wake of the severe trauma to cord and bone, regardless of the operative procedure employed.

It is a well-known fact that the higher the lesion in the cord and the greater its extent, the graver the prognosis.

I am happy to relate that in recent years, due to earlier, better and more accurate diagnosis and treatment, the prognosis in spinal column injuries is more favorable. This has been brought about by better elucidation of the histories of these cases and by the improved technic of making, taking and interpreting roentgenograms.

Dislocations and fractures of the spine often escape early diagnosis, for unless there is involvement of the spinal cord there may be no early signs or symptoms to attract one's attention to the site of the injury.

Many patients with dislocations or fractures of the spinal column have other serious injuries that are easily recognizable which overshadow the spinal injury, so that it may be unrecognized for days, weeks, and even months.

Then a kyphosis, a stiff neck or spastic back will call attention to the undiagnosed, crushed, and dislocated vertebral body. Only by the most thorough history and examination at the time of the accident with free and unlimited use of the x-ray (anterior-posterior and lateral views) will these too frequently unrecognized conditions be diagnosed.

I believe it can safely be said that approximately 25 per cent of fractures and dislocations of the spine are not correctly diagnosed during the first week following their occurrence. This is not an indictment of the medical profession, but rather a humiliating confession that may be lessened if the fact be clearly and forcibly brought to the attention of traumatic surgeons. Kummel's fracture (pathological fracture) is the exception.

Extreme flexion of the neck is one of the chief causes of dislocations in the cervical region, and is often produced in the simplest manner. Bumping one's car from behind may cause a dislocation of one of the cervical vertebrae. Being thrown from a car, falling on the head, sudden and forcible twisting of the neck, are some of the more frequent causes. Direct trauma may produce fractures and dislocations, or both. Spontaneous dislocations are due to chronic infections, arthritis, infantile paralysis, and so on, and are not infrequent.

"Hangman's dislocation," axis on atlas, is the most serious of all cervical dislocations. Fracture-dislocation of the odontoid process with pressure on the medulla is also a very serious spinal cord and column injury. In this fracture dislocation, death may be instantaneous or the patient may survive for a few hours. There have been very few cases of recovery recorded following the injury.

Fractures in the lower cervical and upper dorsal regions with cord lesions are less serious. Death from these lesions usually results from hypostatic pneumonia, while lower down in the spinal canal death usually results from infection and exhaustion. Large decubitous ulcers frequently complicate such cases.

"Miner's back fracture" commonly occurs in the dorsolumbar region and is caused by coal or shale which falls upon the flexed back of the miner, jack-knifing him, and producing a forward dislocation of the vertebral body. This dislocation is usually accompanied by a compression fracture of the body and may involve the lamina or spinous process.

Flexion fractures most frequently occur where the fixed parts of the spinal column join the mobile portions, cervicothoracic, dorsolumbar, and lumbosacral. The first lumbar is the most frequently involved of all the vertebrae, while the twelfth dorsal, the second lumbar, the eleventh

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dorsal and the fourth lumbar are next in frequency, in the order named.

The prognosis and treatment of fracture dislocations of the spine is dependent upon three essential factors, as follows: whether the lesion of the spine is a simple bone injury, a cord injury, or a combination of both. Here, as in fractures of the skull, the seriousness of the case depends not upon the extent of the bone lesion, but upon the extent of the injury to the brain tissue and, in this case, to the cord substance.

Fracture dislocations of the spine without damage to the cord, if recognized early and properly treated, are usually of small consequence, but with cord lesion, paralysis and other serious complications, they are of grave concern.

In fracture dislocations of the spine where there is a complete transverse lesion of the cord, motor and sensory paralysis is complete and usually occurs immediately. In partial severance of the cord, some muscles are active and some sensation is present, paralysis is not so immediate; and later on knee reflexes will return.

In transverse severance of the cord, paralysis of the bladder and rectum occur. In partial severance or where there is pressure due to hemorrhage or bony spicules these organs are usually paralyzed, but not always so. Priapism and free sweating usually occur in complete cord severance.

The site of the fracture should be readily recognized by the loss of sensation in the skin and extremities distal to the cord lesion. The Queckenstedt test is of inestimable value in deciding whether the spinal canal is blocked, and it is a substantial aid in the diagnosis of pressure upon the cord whether due to bone or blood clot.

Having established the diagnosis, the question naturally arises of how best to proceed to relieve the condition.

Simple dislocations and fractures in the cervical region without cord lesions can be treated either by the traction - hyperextension method (head halter and weight and cast later) (preferable method) or by the more drastic procedures of Walton and Taylor (immediate manipulation with or without anesthetic and the immediate application of the cast). These procedures are good, but are more dangerous than the gradual traction-hyperextension method.

Some writers say that traction is used only by those who do not know the Walton or Taylor method or by those afraid to use manipulation. There is a place for both procedures, but nearly all cases respond to traction and hyperextension where it is properly and promptly applied. Most of our cases have been accorded this treatment.

Traction is the method of choice in fracture dislocations of the thoracic and lumbar regions along with hyperextension either by the Walton frame or some modification. After a few weeks the patient is put in a cast and after three to six months in a brace for six months or longer. Fractures due to gunshot wounds with neurological symptoms usually require open operation.

Spondylolisthesis and dislocations of the third, fourth and fifth lumbar vertebrae are treated by traction on both lower extremities, alone or associated with manipulation. During manipulation counter traction is made on shoulders by assistants. Some improvement usually follows this manipulation, but perfect reduction is not the rule. Later a cast must be applied. If paralysis is present, immediate application of the cast is imperative to prevent large decubitus ulcers.

Most of the cases of fracture dislocation of the spine occur in working men, railway and corporation employees. The traction - hyperextension method has not only reduced the morbidity, but has enabled these men to return to their former occupations with no or minimum disability. The loss to compensation insurance companies has been greatly reduced thereby, whereas the earning power of the individual has been maintained.

In the past 28 years we have treated at the Newell Clinic 11,143 fractures. Of these there were 255 fractures and dislocations of the spine (2.3 per cent). The majority were fractures. Gunshot fractures are not included.

In this series there were no deaths in those without spinal cord lesions. In those with cord lesion, in the cervical region, we had our highest death rate.

SUMMARY AND CONCLUSION

The treatment of fractures and dislocations of the spine is more satisfactory in recent years than formerly. This is due to better diagnoses, better radiography and to more prompt and better treatment. Many spine injuries are overlooked when first examined. Thorough examinations and repeated x-rays to check the progress should be made.

Our most satisfactory results have been obtained in pure bone lesions, or where bone trauma is complicated by mild cord injuries. Traction

and graduated hyperextension are the methods of choice in the treatment of these conditions. Some cases require more traction and less hyperextension; others *vice versa*.

The transverse cord lesion, where grave trauma has been done to the cord, offers the least hope. Major surgical procedures for accurate diagnosis and treatment, laminectomies, "fixation operations," are demanded in most of the above cases, but the morbidity is still great and the mortality is high. Unless there are definite signs and symptoms of cord compression or injuries and the Queckenstedt test is positive, it is best not to operate.

There has been a very definite trend away from operative procedures in the past few years in most cases. Many cases that were subjected to laminectomy a few years ago are now treated more successfully by traction and hyperextension; yet not all of them can be so treated.

I think Dr. Dandy, of Baltimore, pointed the way many years ago to our present day treatment when he showed conclusively that many brain injuries and skull fractures did better without operation than those operated upon, discriminative surgery, and so it should be with fracture dislocations of the spinal column.

CASE REPORTS

Case 1.—J. M. F., Jr., a white American, aged 9, November 7, 1933, dislocated the second cervical vertebrae by a sudden twist of the neck. Pain and stiffness followed. There were no neurological symptoms. The treatment was traction-hyperextension for two weeks and cast for six weeks. A perfect result was obtained.

Case 2.—M. D., a white American, aged 34, November 7, 1933, was injured by a racing ambulance which hit the rear bumper of his car. A compression fracture of the fifth cervical vertebra followed, with pain and spasticity, but no neurological symptoms. Treatment was hyperextension with traction for three weeks, and plaster cast for three months. The result was perfect function, with no pain.

Case 3.—J. M. P., a white American, aged 25, was in an automobile accident October 28, 1936. Dislocation of the sixth on the seventh cervical vertebra followed, and chip fracture of the seventh cervical vertebra. Treatment was traction-hyperextension. There were no neurological symptoms. Perfect anatomical and good functional results were obtained. The patient is under treatment.

Case 4.—R. H., a white American, aged 32, in a railway accident August 2, 1936, had the right arm cut off by the wheel of a box car. He did not notice pain or discomfort in the neck or back for two or three weeks. There was dislocation of the seventh on the first thoracic vertebra, and fracture of the sixth cervical, also compression fracture on the right side of the second lumbar vertebra and transverse processes. There was a fractured spinous process of the sixth cervical vertebra. Hyperextension was used for three weeks, cast for six months. The patient is under treatment. So far a splendid result has been obtained. There is no pain nor discomfort. None of these cervical cases so far have required braces after removal of the cast.

Case 5.—R. H. A., a white American, aged 27, on February 2, 1934, was crushed in the mines and suffered a compression fracture of the first lumbar vertebra. Paralysis from the waist down included the bladder and rectum. Treatment consisted of hyperextension on a modified Walton frame for three weeks. There was no improvement in pain or neurological symptoms. Operation was undertaken. Laminectomy was done with removal of blood clots and a spicule of bone pressing on the cord. Hyperextension in a plaster shell was then employed. The end result was almost perfect. He drags the right toe a little when he walks.

Case 6.—R. R., a white American, aged 34, fell from a telephone post May 11, 1934, landing on the buttocks. There were fractures of the twelfth thoracic vertebra. Treatment consisted of hyperextension by padded ten-inch sand bag for one month, followed by hyperextension in plaster cast for three months. Perfect functional and anatomical results followed. There were no neurological symptoms at any time. He returned to his original work.

Case 7.—P. R., a white American, aged 42, on August 29, 1936, was crushed in the mines by a falling roof with

Fig. 1, Case 7

Showing marked lateral dislocation (to the right) of the fourth lumbar vertebra on the fifth with the right lateral one-fourth and anterior portion of the body of the fifth lumbar vertebra being fractured off. Also fracture of the left transverse process of the second and third lumbar vertebrae.

Fig. 2, Case 7
Showing reduction that was obtained by manual manipulation, under general anesthesia.

Fig. 3, Case 7
Showing application of cast. (Patient now walking with a cane.)

lateral dislocation of the fourth on the fifth lumbar vertebra and comminuted compression fracture of the fifth lumbar. There was paralysis of the bladder, rectum and extremities below the knees. Manual manipulation accomplished 85 per cent reduction. Paralysis was partially relieved in a week. Paralysis of the bladder and rectum was entirely relieved in two and one-half weeks. The patient was dismissed with a cast from the shoulders to just above the knees. He is greatly improved and is still under treatment.

Case 8.—In October, 1926, J. H., a Negro American, aged 32, was run down by a train. There was complete lateral displacement of the twelfth dorsal on the first lumbar. I was unable to replace it under anesthesia. There was a transverse lesion of the cord, with complete paralysis below the lesion. He was treated on a Bradford frame with extension. He died three months later from sepsis and exhaustion.

Case 9.—On September 2, 1936, C. C., a white American, aged 38, was in an auto accident. He spent one night in jail and traveled 150 miles two days later by rail before presenting himself for treatment. He did not suspect injury to his spine. X-ray revealed compression fracture of the second lumbar vertebra. The only physical signs that the patient had was spastic back in the lumbar region. Treatment consisted of hyperextension in plaster cast. He was permitted to return to his home in Alabama on the tenth day. He walked out, said he had no pain, and x-rays showed decided improvement in the compression fracture. He is under treatment.

Case 10.—July 3, 1935, A. B., a white American, aged 45, was seen. He had been in a recent auto accident. He has spondylolisthesis. This may be congenital and activated by the injury. Treatment was by manipulation and traction. There was slight improvement. Later a brace was applied. Pain was much less when he was last seen. Most cases are congenital and present few neurological manifestations.

DISCUSSION (*Abstract*)

Dr. Francis J. Kirby, Baltimore, Md.—Fractures and dislocations of the vertebral column call for close, keen observation and early care.

There is probably no class of fractures and dislocations which, when not recognized early and properly handled, results in such pain, discomfort and unfitness for the pursuit of activities, coupled with indefinite suffering, as do lesions of the vertebral column. They are likely to remain a source of much concern to the physician.

Skillful treatment, even in some late cases, has accomplished remarkable results with restoration of function and freedom from pain and muscle spasm.

In fractures and dislocations or both of the bony coverings of the central nervous system, the brain and the spinal cord, the bone injury may be less dangerous than the damage to the central nervous system and its appendages, which results in prolonged discomfort, pain, loss of function from partial or complete paralysis, or in death.

Agreement is general that the essential treatment is rest: early rest with the patient in such a position that pressure and tension are removed from the injured area, and provision is made for mechanical or natural reduction and adjustment of fractured surfaces, so that a close return to the former anatomical and physiological functions may result.

This requires that the head, vertebral column and pelvis be put at rest until nature effects her repairs. If dislocation is the main factor, an attempt at reduction should be made, whether paralysis is present or not (Taylor method).

Dislocation or fracture of the cervical spine calls for most careful attention and watchfulness both by the physician and attendants. These cases should be given care without delay.

X-ray examinations (through the open mouth in cervical lesions) both antero-posterior, lateral and oblique if necessary, should be made in all cases frequently, especially in the lower cervical, dorsal and lumbar regions, since lateral views demonstrate a lesion which the antero-posterior view does not.

Mass reflex, or automatic motion as it has been called, comes on in two or three weeks and is not an evidence of recovery of structures and return to function, but rather that function will be long postponed or never return. In dorsal vertebrae dislocation, at times due to the rebound of the bony thorax, the dislocation is reduced but severance of the spinal cord occurs.

In lumbar dislocations owing to the strong fixation of parts, reduction is impossible.

The history of injury, pain in the back at the point of injury, partial or complete paralysis below the level of injury, are sufficient for diagnosis of fracture in most cases.

The non-operative treatment, which has given the best results in the majority of cases, is hyperextension of the vertebral column, with due care to the prevention of bed sores and irritation. Attention must be given the bladder and rectum for the avoidance of sepsis.

U. J. Mixter has very clearly outlined indications for operative and non-operative treatment as follows. Early cases with progressive neurologic symptoms should have laminectomy. Stationary neurologic symptoms with block should have laminectomy. With diminishing neurologic symptoms, no operation should be done, and with no neurologic symptoms, no operation.

Old cases with increasing signs should have laminectomy. With stationary or diminishing signs and no paralysis, there should be no operation.

While according to Doub nearly one-half of spine fractures are confined to the lower dorsal and upper lumbar regions, there is left a large percentage for fractures and dislocations of the cervical and upper dorsal areas.

Where the spinal cord has been very badly crushed, or cut across, the prognosis with any kind of treatment is practically hopeless.

E. Hauser says that spastic paralysis due to a compression myelitis in which an interval has elapsed between the time of injury and the development of the paralysis can be cured by hyperextension. He cites several cases.

According to E. Vance, of Australia, dorsiflexion is the backbone of treatment of all grave and doubtful cases of injuries of the spinal column. Let us adopt it and more patients with grave injuries will recover, and the less severely injured will be spared disabling kyphosis and mechanically weak backs.

Dr. Newell (closing).—My reason for writing this paper was to call attention to a rather new approach to a problem that in the past has had a very unfavorable prognosis, but which is now handled with a great deal more satisfaction to the traumatic surgeon and comfort to the patient.

HERNIATION OR RUPTURE OF THE INTERVERTEBRAL DISC

A REPORT OF TWO CASES*

By R. F. SLAUGHTER, M.D.
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Since 1911 when Goldthwaite[1] in this country and Middleton and Teachers[2] in England first described rupture and extrusion of the intervertebral disc into the spinal canal very little was said about this condition until about 1916. Since then there have been several articles written on the subject.[3 4 5 6 7 8 9 10 11 12] Peet and Echols[13] gave a very good account of the condition and a report of two cases observed at operation. Mauric,[14] in 1933, concluded from a study of the literature that many of the cases of enchondromata and Schmidt's nodules were identical. Mixter and Ayer,[15] in 1935, reported a series of thirty-four cases proven at operation, twenty-three of which were in the lumbosacral region. From their study they arrived at the conclusion that these cases represented a true rupture of the annular ligament and a protrusion or herniation of the nucleus pulposus into the vertebral canal with pressure against the nerve root and the contents of the dura. The close cooperation of the orthopedic service and the neurosurgical service in patients with seemingly incurable low back pain with or without sciatica produced this remarkable collection of cases. Such a cooperation as this represents medicine at its best.

It is the purpose of this paper to present two cases which were proven at operation and completely relieved of a disability that had been designated as permanent.

REPORT OF CASES

Mrs. R. D. (N.G.H. 24, 40), a white female, aged 36, complained of pain in lower right back and leg since the birth of her child eight years before. She had no complaint previous to her delivery. Following labor she was weak in her right leg and had severe pain radiating down this limb for which she had to take morphine in large quantities from May until December. After December she attempted to walk but still complained of pain in her right leg at times and a constant low backache. She remained weak in the leg for some time but this gradually improved. At the time she was admitted to the hospital she complained of pain in the right leg with a feeling of numbness. At times at

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*From the Neurological and Neurosurgical Service of the Norfolk General Hospital.

night she would have a feeling of pins and needles with cramps. She had some difficulty controlling her urine.

Neurological examination disclosed an over-anxious woman who appeared not to be of the neurotic type but quite desirous of getting relief, although she had been to many physicians. The motor system presented a slight foot drop noticeable on walking and a weakness of the calf muscles. There was subjective numbness in the lower leg in the peroneal distribution with an objective diminution in touch and pin prick in the right great toe. The reflexes disclosed an absence of the right ankle jerk with an increase in the knee jerk on the right as compared with the left. There were no pathological reflexes. The sympathetic system was normal as well as the endocrine. A diagnosis of an extradural cord lesion was made and the patient referred to the hospital for further study.

On admission to the hospital, lumbar puncture was done which showed an increase in protein as determined by the Pandy test. The spinal fluid showed no evidence of block and the further examination of the fluid was negative. In such a case it is my policy to introduce 5 c. c. of iodized oil into the spinal canal of the patient on a tilting table and under the fluoroscope to observe the progress of the opaque medium up and down the canal. This will disclose any defect that may be present, by a filling defect in the column of oil or a blockage of its descent. When the defect is found a flat plate is taken for permanent record. In this case, because of a delay in getting the iodized oil, air was used as a contrasting medium. No defect was found. The findings were so significant, however, that I thought a laminectomy was indicated. The third, fourth and fifth lumbar laminae were removed in the classical manner and extending around the dura, markedly compressing it was a fibrocartilagionous protrusion at the level of the fourth to fifth lumbar. This was removed extradurally and a clamp was introduced into the cavity left by the mass, which cavity was found to descend into the depth of the intervertebral space. A diagnosis was therefore made of a herniated disc. Following the operation there was an immediate improvement. When the patient left the hospital there was a return of strength in the leg and a loss of all pain, the first time in eight years. Follow-up examinations have proven that she is entirely relieved.

Case 2.—Mr. W. A. B. (N.G.H. 3884), a 69 year old male laborer, was admitted to the Norfolk General Hospital on August 3, 1936, with a complaint of pain in the lower back radiating down the right leg with limitation of motion of the right leg.

On May 13, 1936, he stepped on a rock while carrying a heavy load and fell, striking his right hip and twisting his back. There was immediate onset of pain in the lower back radiating down the right leg. For three weeks he was confined to bed, but the pain persisted in spite of strapping and radiation of heat. On attempting to walk he discovered that bending the leg caused severe pain down the course of the sciatic nerve and walking up the steps was impossible because of pain. He also noticed that straining for a bowel movement or coughing or sneezing caused pain to radiate down the back of his leg. He had been treated for arthritis. His pain and disability continued and the patient was referred to me by Dr Mallory Andrews for an opinion.

Upon examination the patient walked with a cane, leaning forward heavily upon the cane. The leg was swung in a rotary fashion, the patient being careful not to bend it and taking short steps. The pain made him rest frequently. A general physical survey disclosed a slight enlargement of the heart with a blood pressure of 178/122. There were no murmurs. There was a moderate degree of arteriosclerosis. Lungs, abdomen and genitalia were normal. There was extreme pain on flexing the right leg and extending it. There was pain on deep pressure over the sciatic notch and along the sciatic nerve. Flexion and extension of the left leg caused no trouble.

Neurological examination revealed a mentally agitated patient willing to accept any treatment that would relieve his pain. The cranium was negative to palpation and auscultation. The cranial nerves were negative. The motor system showed the disability described above. There was no atrophy, abnormal movement nor asynergia. There was weakness in the right leg due to splinting of the muscles caused by pain. The sensory system disclosed hyperesthesia in the right leg as compared to the left. This may have been due to the mental effect of constant pain. The knee jerk and ankle jerk were more active on the right as compared with the left. The sympathetic and endocrine systems appeared normal.

A lumbar puncture was done between the fifth lumbar and the sacrum. The initial pressure was 170 mm. of water. There was a good rise and fall on coughing and straining. Jugular compression gave a steady rise and fall to 300 mm. of water returning to 170 mm. of water. Coughing and jugular compression caused pain to radiate down the right leg. Ten c.c. of fluid were removed and the procedure repeated from the initial level of 100 mm. of water. There was a rapid rise to 250 but the fall returned to 130 to 140 mm. of water. This was thought to be significant but not diagnostic. The laboratory reported a spinal fluid protein of 64 mg. with a marked increase in the Pandy test. There were 33 cells and a midzone rise in the colloidal gold test. The blood and spinal fluid Wassermann tests were negative.

Five cubic centimeters of heavy iodized oil was injected into the subarachnoid space at lumbar 1-2 and its descent watched under the fluoroscope. There was a constant "nicking-filling-defect" on the right side at lumbar 4-5. Spot films were taken showing the defect.

Following the injection there was a marked increase in the pain requiring an increase in the frequency of codeine which he had been taking since the Fall. He was operated upon on August 27, 1936, under gas and ether. (Spinal anesthesia was contemplated because of his age and condition). The laminae of the 4-5 lumbar vertebrae were removed and the dura rotated to the left disclosing a rounded, glistening nodule the size of the end of the little finger presenting between the dura and the nerve root. The nerve root was displaced, congested and swollen. The nodule was removed with care to prevent injury to the nerve root and a clamp was introduced into the socket whence it came, proving its course. The dura was opened and the iodized oil evacuated.

Following the operation there was immediate relief. Complete relief of pain did not occur until three weeks following operation which allowed time for the neuritis to subside.

DISCUSSION

From the operative and pathologic findings I am of the opinion that these cases represent true rupture of the annulus and extrusion of the nucleus pulposus. A history of trauma was present in both patients. Low backache with sciatica-like pain, which was disabling, caused both patients to present themselves for treatment. Reflex changes occurred in the extremity in which there was a complaint of pain. Muscular atrophy and sensory changes were found in the case which had a history extending back eight years. Both patients complained of pain on sneezing and coughing which radiated down the posterior aspect of the leg. Sphincter disturbance was present in one patient. On lumbar puncture there was an increase in protein in both cases with no spinal block. Roentgen studies after injection of iodized oil were made in one case which demonstrated a typical filling defect. Operation upon both cases disclosed the lesion and its source. Complete recovery followed both operations as was proven by the follow-up examinations.

Fig. 1
Rounded filling defect resembling tumor outlined by the oil.

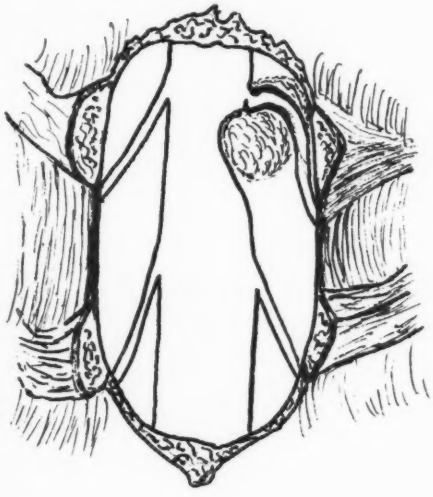

Fig. 2
Operative sketch of lesion. Note the swollen root with displacement.

Fig. 3
Diagrammatic sketch of herniated disc.

If a patient is seen with the history that he has had a wrenching or twisting of his back in a fall or a "jack-knifing" of his back which was immediately followed by pain in the lower back radiating down the leg or legs, and this pain has persisted in spite of absolute rest in bed, it is well to bear in mind that this patient may have a ruptured intervertebral disc. If, however, upon repeated examinations one finds that the clinical picture changes frequently the pa-

tient may have a functional disability. If rest in bed completely relieves his complaint and motion brings it back, then orthopedic treatment is indicated. These statements are, of course, relative.

The injection of iodized oil will disclose the presence of the defect in every case and laminectomy should not be contemplated in its absence. The technic consists of injecting 5 c.c. of iodized oil into the first lumbar interspace and by means of the tilt table the oil is observed under the fluorscope as it descends to fill the caudal sack. The patient is placed in various positions as the oil flows back and forth and when the defect is seen spot films are taken as a permanent record. A trained observer is needed, however, to determine whether the defect is artificial or real. The suggestive history and the presence of an increase in protein in the spinal fluid is the indication for this study.

A review of the literature indicates that the lumbar spine is the most vulnerable for this affliction, 42 cases having been reported. In the lumbar spine the 4-5 interspace is the most frequent site. Twelve cases have been reported in the cervical region. These patients present the typical findings and symptomatology of a cord tumor. In seven of the eight cases reported by Mixter a spinal block was demonstrated as were the usual spinal fluid findings. At operation the lesion presents itself in the midline and rotation of the cord by the dentate ligament is necessary for its removal. The eight cases reported in the literature did not show as good a result as the lumbar cases after operation. The reason for this is probably due to the defect left in the intervertebral space after the extrusion of the disc, or possibly further herniation of the remaining disc. A small lesion will produce major difficulties in this region. Spasticity was a constant finding in these cases.

There were 27 cases presenting herniation in the dorsal region. These cases differed from the cervical lesions only in the fact that ataxia was a prominent symptom.

CONCLUSION

Two cases of herniation of the intervertebral disc are presented. The symptomatology and findings are similar to those reported in the literature. The laboratory and x-ray findings are characteristic and diagnostic. Operations were performed upon both cases, at which time the herniation was demonstrated by introducing a clamp into the socket from whence the disc had extruded. Subsequent pathological reports substantiated the diagnosis. Both cases were relieved after operation as reported in the follow-up examinations.

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REFERENCES

1. Goldthwaite, J. E.: The Lumbosacral Articulation: An Explanation of Many Cases of "Lumbago," "Sciatica" and Paraplegia." Boston Med. & Surg. Jour., **164**:365, 1911.
2. Middleton, G. S.; and Teacher, J. H.: Injury of the Spinal Cord Due to Rupture of an Intervertebral Disc During Muscular Effort. Glasgow Med. Jour., **76**:1, 1911.
3. Elsberg, C. A.: (1) Diagnosis and Treatment of Surgical Diseases of the Spinal Cord and Its Membranes, p. 238. Philadelphia and London: W. B. Saunders Co., 1916.
4. Ibid: (2) Extradural Spinal Tumors: Primary, Secondary, Metastatic. Surg., Gyn. & Obst., **46**:1, 1928.
5. Ibid: (3) Extradural Ventral Chondromas (Ecchondrosis); Their Favorite Sites, Spinal Cord and Root Systems They Produce and Their Surgical Treatment. Bull. Neurol. Inst., New York, **1**:350, 1931.
6. Clymes, G.; Mixter, W. J.; and Mella, Hugo: Experience With Spinal Cord Tumors During the Past Ten Years. Arch. Neurol. & Psychiat., **5**:213, 1921.
7. Stookey, B.: Compression of Spinal Cord Due to Ventral Extradural Cervical Chondromas: Diagnosis and Surgical Treatment. Arch. Neurol. & Psychiat., **20**:275, 1928.
8. Bucy, P. C.: Chondromas of Intervertebral Disc. J.A.M.A., **94**:1552. 1930.
9. Petit-Dutaillis, D.; et. Alajouranine, T.: Syndrome unilateral de la queue de cheval, laminactomie exploratrie et ablation d'un fibrome due disque intervertrebral. Bull. et Mem. Coc. Nat. de Chir., **54**:1452, 1928.
10. Adson, A. W.: Diagnosis and Treatment of Spinal Tumors. Northwest Med., **24**:309, 1925.
11. Dandy, W. E.: Loose Cartilage from Intervertebral Disc Simulating Tumor of Spinal Cord. Arch. Surg., **19**:660, 1929.
12. Alpers, B. J.; Grant, F. C.; and Yaskin, J. C.: Chondroma of Intervertrebral Discs. Ann. Surg., **97**:10, 1933.
13. Peet, M. M.; and Echols, D. H.: Herniation of Nucleus Pulposus: Cause of Compression of Spinal Cord. Arch. Neurol. & Psychiat., **32**:924, 1934.
14. Mauric, G.: La Disque Intervertebral: Physiologie, Pathologie, Indications Therapeutiques. Paris: Masson et lie, 1933.
15. Mixter, J. W.; and Ayer, J. B.: Herniation or Rupture of the Intervertebral Disc into the Spinal Canal. New Eng. Jour. Med., **213**:385-393, Aug. 29, 1935.

THE SURGICAL TREATMENT OF LOW BACK PAIN AND SCIATICA*

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We have reviewed the cases of all patients treated at The Mayo Clinic by operations designed to produce bony fusion of the lumbosacral or sacro-iliac joints, or both, and have included in the study those cases in which the operation of excision of a transverse process was done for relief of sciatic pain. We have not included in this series, however, any cases of spondylolisthesis in which treatment was by bone graft or fusion operations, any cases of tuberculosis or compres-

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sion fracture of the vertebral bodies, or any neurosurgical cases.

This troublesome condition has been treated surgically for a good many years. Some of the operations reported in this paper were performed as far back as 1914; most of them, however, have been performed since 1922. That methods of handling the condition from the surgical standpoint have changed considerably is obvious on reviewing these cases. In the earlier cases resection of transverse processes was the operation most often used. Later, fusions and bone grafts to the sacro-iliac joint came into vogue. More recently, lumbosacral fusions, either with or without fusion of the sacro-iliac joints, have been the usual choice of procedure. *Table 1*

Table 1

COMPARISON OF END RESULTS OF OPERATIONS PERFORMED MANY YEARS AGO AND THOSE PERFORMED DURING THE PAST FEW YEARS AT THE CLINIC FOR LOW BACK PAIN AND SCIATICA

Procedure	Cases	Percentage of Good Results Obtained
Removal of transverse processes*	14	28.5
Sacro-iliac fusion*	7	28.6
Lumbosacral graft from iliac crest with sacro-iliac fusion†	47	72

*Early procedures.

†Procedure employed in recent years.

shows the wide difference in end results obtained following operations performed many years ago and those performed in the last few years. It is to be hoped that by further improvement in diagnostic as well as surgical methods these results may be further improved.

Certain facts must be obvious to all concerned with this problem: (1) If fusion of a joint is to relieve pain, that joint must be demonstrated to be the underlying cause of the trouble, a fact which may be hard to demonstrate. Pathologic changes which may cause symptoms in the lumbosacral or sacro-iliac joints are difficult to study because of lack of material. From the studies of various investigators, particularly Schmorl and his associates, we know that the intervertebral discs may be damaged, or the nucleus pulposis ruptured or extended either into one or the other adjacent vertebral bodies or into the spinal canal. Subsequently, fibrosis and atrophy of the disc may produce definite narrowing of its space, as demonstrated in the roentgenograms. With these changes more pressure is exerted on the adjoining intervertebral articulations and the surfaces of facets become the site of changes typical of traumatic arthritis. Such changes may produce symptoms, particularly if repeated strains are exerted on the affected part. (2) The operation must adequately fuse the affected joint or joints if it is to produce relief from symptoms. (3) Exclusion of all other causative factors should be accomplished before operation is attempted. (4) Operative treatment should not be used as a last resort because all other methods have failed. Such a reason is not an indication and operations performed for this reason alone will often lead to an unsatisfactory result. We believe this point to be a most important one in considering the treatment of these patients. Too often the surgeon is more or less driven to radical treatment because of the fact that the patient has not responded to other types of conservative treatment. Such a procedure may be justified, but other indications than this should be found before any such surgical procedure is adopted.

Certain criteria for operation in these cases may be adopted by various surgeons. As far as we are concerned the general indications are as follows: (1) persistent low back pain with or without sciatica, (2) failure of a fair trial of adequate conservative measures, (3) roentgenographic changes, such as narrow lumbosacral disc or facet changes, or both. (We have been unable to establish any roentgenographic criteria to demonstrate changes in the sacro-iliac joint which we believe are of much help in deciding for or against operation. Positive evidence of destructive lesions in these joints may be found, fusion may be demonstrated in properly made stereoroentgenograms, and arthritic changes and para-articular osteitis may all be demonstrated at times, but none of these changes can be taken as criteria for operation. Roentgenograms should be made, but in most instances selection of cases for sacro-iliac fusion will be made largely on other evidence), (4) negative neurologic findings, including lumbar puncture in most cases, (5) consistent findings on physical examination, particularly areas of tenderness; such patients should be examined several times to be sure the findings have been consistent, (6) a static type of pain, and (7) the patient should be between the ages of 20 and 50 years.

In the selection of cases for operation the importance of deciding whether or not to fuse the lumbosacral as well as the sacro-iliac joints cannot be over-emphasized. We do not believe any tests yet available are absolutely reliable in determining which joint is involved. If there is any doubt about this and operation is elected, it is far wiser to fuse both joints rather than

run the risk of failure by fusing one or the other alone. Our tables showing end results substantiate this observation.

Such rules, of course, may not be always followed, yet if they are followed we believe the results will be more uniform and satisfactory. When conservative measures have not been tried, one should first give them a fair trial, if possible, before operation is undertaken. It is our custom at the clinic in most of these cases to place the patient in the hospital for a period of ten days to two weeks. During that time the patients are on a firm bed with a fracture board and hair mattress. Buck's extension is applied to both legs, usually 6 to 8 pounds (2.7 to 3.6 kg.). A lumbar sling, counterweighted to support the lumbar spine, is applied. Physical therapy is usually given daily, baking and massage being alternated with diathermy until it can be determined which is the most beneficial.

Usually the patient is uncomfortable during the first two or three days of such treatment, after which improvement should be looked for. If such improvement does not come, epidural injection may be tried. In a number of cases this will relieve the pain more or less completely. If complete neurologic investigation has not been carried out, including injection of iodized oil to rule out avulsion of a disc, it should be done at this stage provided no relief has come with the treatment already instituted. However, if there is improvement which continues after several days, usually from ten days to two weeks, the patient should be allowed to get up and he should be fitted with a corset or belt. If recurrence of pain comes with resumption of activity, and especially walking, we believe that one should then consider the advisability of operation. If no relief has come within ten days to two weeks on such a program it is usually wise to postpone any surgical treatment for a period of from three to six months and then ask the patient to return for reexamination. In some instances developments may take place which entirely change the clinical picture during such an interval.

We regard static pain as a very suggestive aid in the selection of these cases for operation. If rest will not relieve the pain, it seems unlikely that fixation of a joint will relieve it. In our series of 155 cases "static" pain was the predominant type of pain in ninety-six cases.

We have set the age of 50 years as approximately the upper limit for operation. We feel that the natural changes in the ligaments and joints will produce sufficient fixation within a few years after 50 to alleviate symptoms to a large extent. In the same way we regard severe recurrent backaches in a younger individual as most likely to be operative, other indications being satisfied. Such individuals, if skeletal changes in the roentgenogram exist, are likely to have recurrent episodes of painful backache over a period of many years. In this series the average age at the time of operation was 33 years. The average duration of symptoms, however, was only 5.5 years.

The selection of the type of operation must of course vary with the surgeon and his individual likes and dislikes. There are several adequate operations. Differences in the type of operation are probably important only insofar as the individual surgeon's experience has carried him in the use of such an operation.

In our series of cases two main types of operative procedure have been used in cases of lumbosacral lesions. The first type is that described at the American Medical Association meeting in 1933 in which a bone graft is taken from the crest of the ilium, after preparing a bed for it by denuding the spinous processes and laminae of their ligamentous and muscular attachments and partially resecting the articular facet and removing the remaining cartilage from the surfaces, or in some instances completely resecting the facets; the latter group of cases will be discussed more fully later. In the second type of operative procedure the same sort of preparation of the bed and the use of tibial bone grafts, usually placing one on either side and packing numerous chips of cancellous bone around them, is used. With either of these operations fusion of one or both sacro-iliac joints may be carried out. Both the Smith-Petersen and Campbell types of sacro-iliac fusion have been used in conjunction with lumbosacral fusion.

The first type of operation is adequate when one wishes to ankylose only the fifth lumbar vertebra to the sacrum. The second type is necessary when more than the fifth lumbar vertebra is to be ankylosed to the sacrum or when a bifid sacrum is found necessitating more bony graft to obtain contact.

In the earlier cases of sacro-iliac fusion alone a graft from the tibia was used which extended from the posterior superior spine to the sacrum. Following the report of Smith-Petersen's operation it was used more frequently, and later Campbell's operation was performed in a number of cases. The most commonly used, however, has been Smith-Petersen's operation.

Postoperative care has been fairly uniform. Immediately after operation a scultetus binder is applied and the patient is placed on a Bradford frame in the prone position. This position is usually maintained for a period of twenty-four hours, after which a regular turning schedule is begun, the patient being turned by nurses and orderlies once every six to eight hours. This program may have to be varied somewhat to suit individual cases, some patients being more comfortable on their backs and others more comfortable in the prone position. Such a schedule is maintained for five weeks, after which the patients are permitted to turn themselves. At the end of six weeks they are allowed to sit up with a supporting belt or corset, and usually within a week they have gained enough strength to permit them to go home, but on a very limited schedule of activity; usually they are allowed up only two to three hours a day for six weeks more, but after this they may be permitted to gradually resume their normal activity. At the end of four months roentgenograms are taken. If these show sufficient union between the graft and host bone or in the area of sacro-iliac fusion the patient is allowed to continue his normal activity. At this stage exercises and massage to rehabilitate the atrophied and scarred muscles should be outlined. Swimming and sun bathing are most helpful, but if they are not available in their stead are prescribed exercises to develop the gluteal as well as the erector spinae muscles.

We know that many patients with long-standing disability will not be fully recovered at the end of four months. That amount of time is necessary for fairly good union of the graft to take place. Complete union of the graft may take longer, in many cases as much time as a year elapsing before the maximal amount of new bone is produced. After union of the graft or fused joint is fairly complete the process of rehabilitation and reestablishment of muscle strength may go on for months. It is difficult at times to make patients realize the importance of this phase and a good deal of encouragement on the part of the physician may be necessary.

The accompanying tables are designed to show the results from the various types of operations. In *Table 2* the results of the combined operation of lumbosacral and sacro-iliac fusion have been listed. Good results were considered to have been obtained when patients regarded themselves as entirely free of symptoms referable to the lumbosacral or sacro-iliac lesions. Fair results were those obtained when the patient showed some relief, but still had symptoms. The one death in the group was due to pulmonary embolism in the third week of an otherwise normal convalescence. The failure in the case of lumbosacral graft from the iliac crest with Campbell's sacro-iliac fusion occurred in a compensation case in which the patient was found to have avulsion of a disc.

In *Table 3* the results of lumbosacral graft are shown, and it might be noted that the two types of operation have produced about similar results. *Tables 4* and *5* show the results of sacro-iliac fusion. One may well interpret the lower percentage of good results as indicating that in some of these cases lumbosacral fusion should have been done in addition to sacro-iliac fusion. The unsatisfactory results in the older types of operation is obvious. The same may be noted about resection of transverse processes.

A compensation neurosis may play an important part in some of the failures, and careful selection of cases for operation is probably the only way to avoid such failures. On the other hand, we have obtained some excellent results in compensation cases. It is our belief that before operation is undertaken in most instances,

Table 2

RESULTS OF COMBINED OPERATIONS

	Total Cases	Results									
		Good		Fair		Poor		Dead		Unknown	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Patients	Per Cent	Cases	Per Cent
Lumbosacral graft from iliac crest with Smith-Petersen type of sacro-iliac fusion	47	34	72	9	19	2	4	1	2	1	2
Lumbosacral graft from tibia with sacro-iliac fusion	9	4	45	3	33	2	22				
Lumbosacral graft from iliac crest with Campbell sacro-iliac fusion	1					1	100				

Table 3

RESULTS OF LUMBOSACRAL GRAFT

	Cases	Results							
		Good		Fair		Poor		Unknown	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Lumbosacral grafts from iliac crest	19	11	56.5	3	17	3	17	2	12
Lumbosacral grafts from tibia	15	10	66	4	27	1	6.6		

Table 4

RESULTS OF SACRO-ILIAC FUSION

	Cases	Results							
		Good		Fair		Poor		Unknown	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Smith-Petersen type	36	21	58	8	22	5	14	2	5.5
Campbell type	9	5	55	3	33	1	11		
Other types (mostly "strut" grafts)	7	2	29	4	57	1	14		

Table 5

RESULTS OF RESECTION OF SACRALIZED TRANSVERSE PROCESSES

	Cases	Results							
		Good		Fair		Poor		Unknown	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Resection of sacralized transverse processes	14	4	29	2	14	6	43	2	14

settlement of all compensation claims should be made on a basis of partial or total permanent disability. This will place the patient on his own, as it were, and give the physician an opportunity to work with him for the best result.

We have not reviewed our cases in which fasciotomy was performed because they are too few in number and the patients operated upon too recently to estimate the real value of the procedure.

Another condition of increasing interest is avulsion of the intervertebral disc, and the condition is undoubtedly present more frequently than was previously believed. The use of iodized oil as a diagnostic measure will reveal a higher percentage of such cases. Whether excision of the protruding portion of the disc by laminectomy alone will cure it is as yet not proven.

We have resected facets in many of our cases. In all but one case lumbosacral fusion has been done, either previously or at the same time as the removal of the facet. In all but one of these cases a good result was obtained. In three cases in which resection of facets was done after lumbosacral fusion, the relief of pain has been complete, which strongly suggests that the facet was causing the pain. We feel that the complete removal of the facet is a difficult procedure. There is room for improvement in the technic of this operation.

SUMMARY AND CONCLUSIONS

We believe our review of these cases has shown that a fairly high percentage of cures can be obtained by fusion of the lumbosacral or sacroiliac joints, or both. More accurate diagnostic methods are proving of greater help in selecting cases for operation.

In general, patients whose pain is relieved by rest are the ones who will be benefited by a fusion operation. Resection of transverse processes of sacralized lumbar or lumbarized sacral vertebrae has not in our experience proven to be of great benefit. Removal of facets in some cases is essential to complete recovery.

THE TREATMENT OF PROCIDENTIA UTERI BY THE VAGINAL ROUTE*

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The present treatment of prolapse of the uterus, like all progress in the evolution of the science of surgery, is the sum of the accumulated experience of many years, many countries and numerous illustrious names. Vaginal celiotomy, because of its safety, was the favorite approach prior to the onset of the Lister period, but because of its employment when contraindications existed and of the impunity with which abdominal section could be performed, it fell into undeserved disrepute. Gynecology, too, was in the hands of the general surgeon, who disdained obstetrical practice and therefore could not be expected to acquire such thorough knowledge of the details of the vagino-pelvic anatomy as of the intra-abdominal and intra-pelvic regions. These he viewed from a good sized abdominal incision which could be easily enlarged without cutting a hole in the bladder or severing the ureters. The pendulum has started again in the opposite direction, as evidenced by the trend in the current literature, but most cases of uterine prolapse are still treated abdominally, although the general surgeon is not saying much about it. Therefore, an observer at the different clinics in this country and abroad is confronted by conflicting practices and the careful student of available literature is confused by opposing opinions, not only as to the treatment but also as to the etiology, anatomy and physiology of the structures involved and is disturbed by unsound controversial discussions.

In uncomplicated prolapse of the uterus of the common type, abdominal section is, in our opinion, never indicated nor justified except in congenital prolapse in virgins or nulliparas or as a palliative gesture in child-bearing women in whom sterilization is improper or undesirable. Congenital prolapse is so uncertain of relief that even the unphysiological and unanatomical abdominal fascial suspension by the Kocher-Murphy operation or other similar method with or without obliteration of the cul-de-sac of Douglas by the Moschovitz technic, combined with suitable plastic procedures, is sometimes indicated, in spite of the fact that it is attended by a much higher morbidity and mortality. Vaginal operations for prolapse, in order to be successful, almost always demand that pregnancy be interdicted, therefore palliative relief is often imperative in child-bearing women and can best be afforded by a combined abdominal suspension and plastic operation. It is not within the scope of this paper to enumerate the numerous methods advocated for uterine displacements in this type of patient, but frequent failure of conservative measures bears eloquent witness to the efficacy of a more radical and direct attack as applied to the lesion in women in whom pregnancy and labor can be ignored. These two types of prolapse, if simple retroversion and a relaxed perineum without prolapse are eliminated, represent a small percentage of the total number presenting themselves for treatment, but in them the abdominal route must be given precedence even though recurrence is not only possible but probable. Of course, the presence of large tumors of the uterus or adnexa or extensive inflammatory adhesions may make the vaginal approach hazardous, under which circumstances another technic may be chosen.

An accurate knowledge of the applied anatomy of the pelvis and perineum is of paramount importance in the correct performance of any operation, but argument as to the function of different structural fasciae and muscles is illogical and inconclusive. For practical purposes, prolapse of the uterus is not only prolapse of this organ itself, but also prolapse or stretching of the bladder, rectum, ovaries, ligaments, fasciae, muscles and so forth of varying degree, therefore the treatment must be directed either toward correction of the abnormal relationship of these to other adjacent organs and tissues with the restoration

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of normal function, or toward substitution of an unanatomical but physiological relationship which will tend to correct the abnormal mechanism.

According to Prof. Halban, of Vienna, who has made elaborate macroscopical and microscopical studies of the pelvic and perineal regions, the endopelvic fascia is the chief support of the uterus and is particularly strong in the sub-cervical, vesico-vaginal and recto-vaginal layers, but his clinic finally concludes that the perineum is the natural barrier to prolapse. In opposition to this view, Goff cites the work of Tandler and Halban but agrees with the theories of Alexandroff and the Manchester group that the endopelvic fascia is of value only at its reflection from the cervix (transverse cervical or cardinal ligaments). He repairs the perineum in passing, although he does not believe that prolapse is, in any way, dependent upon the integrity of the pelvic floor. It is the opinion of the essayist that no one or two theories as to the mechanism of prolapse will explain the cause or simplify the treatment, but that one must utilize the structures at hand regardless of the term applied. If the endopelvic fasciae, uteropelvic ligament, muscular and fascial planes of the bladder, rectum or vagina or any other named tissue are well developed, suture them for strength, but searching through attenuated tissues for hidden treasure does not often succeed.

Most authorities agree, however, that procidentia is a form of hernia, generally due to the trauma of labor and that a predisposition to the hernia must be present just as the peritoneal pouch predisposes to inguinal hernia, and that continued deflection of intra-abdominal pressure toward the vulvar cleft is the exciting cause. After careful study of our series, we are in complete accord with these views.

The etiology of prolapse with the exception of congenital anomaly lies almost wholly within the conduct of labor and as the traumatic cause may occur at the first or tenth labor, parity is of slight consequence beyond the natural progression over a period of years. The age of the patient is likewise of little significance in so far as the degree of the prolapse is concerned, as some women bear children at 16 and others at 40. The prophylaxis of prolapse is in the domain of obstetrical practice and chance, but we are far from the time when all labors will be properly conducted and an effete civilization has eliminated chance.

No apology is offered for the omission of a classification for cystocele or rectocele, as the grade of each cannot be accurately determined by a given formula, but must be interpreted at operation. The division of first, second and third degree prolapse is likewise arbitrary and unsatisfactory in that one frequently merges into the other and is largely influenced by the extent of the cystocele or rectocele or the presence of an enterocele. The treatment, however, must in each individual case take due cognizance of all the grade of prolapse of the uterus, as well as of adjacent organs and structures and the importance of each must be correctly evaluated in the application of remedial measures.

As in all lines of surgical endeavor, stereotyped principles have no place in the solution of these problems, but are of necessity modified and influenced by numerous factors, each of which must be carefully considered in order to secure satisfactory results in a large proportion of cases. While fortunately a majority can be cured by a carefully performed operation of almost any accepted method, yet a critical number yield poor results or carry a more disabling lesion than the original one. It also would appear that the multitudinous operations advised have been unfortunately influenced by the methods in vogue in different sections, whether the preceptors are general surgeons or gynecologists, the number of skillful and well trained obstetricians, the custom in many states of midwife practice, the economic and industrial status, general type of inhabitants in certain areas, and many other factors which may, but usually should not, justify employment of a particular operation to the exclusion of all others.

B. H. Goff[1] reports 116 cases with 98.9 per cent satisfactory results in the Bissell operation of vaginal hysterectomy in which the Ward method of cul-de-sac closure is used. The broad ligaments are not interposed in his series, but judging from the technic advised as well as personal observation, no objection can be seen to its employment.

Counselor and Stacy[2] reported 819 cases of prolapse in which 576 vaginal hysterectomies were done with 96 plus per cent good or fair results, but no mention was made of the exercise of any particular care in the closure of the posterior pouch, nor did the writer note any particular emphasis upon its employment at the Mayo Clinic.

The Watkins - Wertheim interposition is de-

scribed as the favorite at Johns Hopkins,[3] Everett finding perfect or satisfactory end results in 96 per cent, while only 80 per cent were given relief by vaginal hysterectomy. Baer and Reis[4] had 91 cases in which the Watkins operation was done with 70 per cent satisfactory results.

Coventry and Moe[5] cured 98 per cent in 70 Watkins interposition cases and 84 per cent in the Mayo hysterectomies done on 30 cases. All recurrences in the latter were in the vaginal vault.

Cattell and Swinton[6] present 76 out of 100 interpositions followed up with 84 per cent good results after 1 to 10 years.

Shaw,[7] from Manchester, reported 549 cases with 96 plus per cent as cured by the Donald-Fothergill-Shaw method, or as it is usually termed the Manchester operation, but we must confess that we are not impressed by the comparatively simple technic employed and the deductions advanced as applied to the usual prolapse seen in our section. The Manchester operation as practiced by Fothergill and others is unbelievably popular in England and English statistics, to which reference has been made, are almost too good to be true. The deductions drawn from these statistical records have not been generally accepted in this country and can probably be explained by a difference in our index of cure and national physical type. It seems feasible to us that the Manchester operation should be placed in the same category as a simple anterior colporrhaphy with pelvic floor repair as recommended for milder grades of prolapse. We are reluctant to barter the apparent more positive interposition of the fundus or broad ligaments for a procedure which is based on the principle of strong para-cervical and paracolpial fascia. The argument often advanced that the Manchester operation can be done during the child-bearing age is unconvincing, since the objective is anteflexion of the uterus, which usually necessitates a high amputation of the cervix and is almost synonymous with sterilization.

The treatment of procidentia is preferably by the vaginal route and varies with the extent of the prolapse. For the simplest type, an anterior colporrhaphy with or without amputation of the cervix and a careful perineorrhaphy may suffice. The bladder may be advanced and, if identified, the utero-pubic ligament separated and sutured in front of the bladder as an adjunct. For the so-called second degree, particularly if the cystocele predominates and the uterus is of the proper size, interposition of the fundus as recommended by Watkins is the method of choice. In the more advanced or the third degree, or if it is necessary to remove the uterus in the second degree, a vaginal hysterectomy with interposition of the united broad ligaments by the method of Mayo has given uniformly good results. Particular emphasis must be placed upon the careful performance of a perineorrhaphy and posterior colporrhaphy as advocated by Kelly many years ago and the high ligation of the sac in enterocele, if present, must not be overlooked. The closure of the cul-de-sac by suture of the utero-sacral ligaments and their fixation to the resected edge of the posterior vaginal wall as recommended by Ward has almost entirely eliminated the vault prolapse with rectocele in vaginal hysterectomies.

The colpocleisis of LeFort, resurrected by Neugenbauer and others, also has a definite place in the treatment of prolapse in elderly women, but its field is greatly restricted.

The operations of choice in prolapse are the modified interposition of Watkins and the modified Mayo vaginal hysterectomy based upon the same principles, one of which will be found applicable in the great majority of cases of procidentia uteri. While general surgery often bears the label of the continental surgeons, whose laborious investigations and detailed descriptions are classical within the field of gastric and colon resections and anastomoses though often modified and improved by American surgeons, gynecology bears the unmistakable imprint of American skill and ingenuity as evidenced by the work of Sims, Emmett, J. R. Goffe, Watkins, Noble, Moore and many other dead as well as living gynecologists who have ably contributed to the art. These two operations have been developed and popularized by the gynecologists of this country, but the contributions of Shauta, Wertheim, Mackenrodt and Duhrssen are basic and have been accorded international recognition.

Various supplementary steps which should not be considered as surgical entities but as adjuncts often account for the difference between success and failure. A carefully and skillfully performed perineorrhaphy is the bulwark in both of them; a fixation stitch which attaches the uterine cornua to the periosteum just beneath the descending ramus of the pubis about two centimeters lateral to the urethra on each side should never be omitted in the interposition of the fundus and is often of value when passed through the round ligament in the same manner after a

vaginal hysterectomy. Kelly's rectopexy or his suture of the fascial bands and the bladder sphincter at the neck for incontinence are often necessary steps, and indications for either should never be overlooked. Ward's suture of the utero-sacral ligaments with closure of the posterior cul-de-sac for enterocele and as a fixation maneuver in the vaginal hysterectomy is of incalculable value, effectively welding the weakest link in the Mayo vaginal hysterectomy.

In practice, we have approached each case with a definite plan in mind, but have attempted to apply the proper operation to the individual rather than the individual to the operation. As a general rule, however, the interposition is done if the uterus is large enough to fill the hernial defect without danger of pressure symptoms and no indication for its removal is present, and vaginal hysterectomy by the modified Mayo technic if the uterus is atrophic or other indications for hysterectomy present themselves. The uterine fundus is always anchored to the resistant tissues beneath the descending rami of the pubes in the interposition and Ward's method or a modification is invariably used in the closure of the cul-de-sac in the vaginal hysterectomy. Of the cases in our series, 52 have been carefully examined after a period of 2 to 15 years with the findings depicted in the slides. We have not resorted to questionnaires in that an individual patient often cannot accurately describe a recurrence in so far as a good result is concerned.

Nowhere in gynecology is careful attention to detail and infinite patience in the application of postoperative treatment of more importance than in the treatment of procidentia uteri. Accurate approximation of tissue and meticulous care is the price of success.

SUMMARY

(1) Vaginal hysterectomy with interposition of the broad ligaments (modified Mayo operation) or transposition of the fundus (modified Watkins operation) are applicable to most cases of second and third degree prolapse of the uterus.

(2) Abdominal fixation and fascial suspensions with the proper plastic procedures, including obliteration of the cul-de-sac of Douglas with sterilization, if necessary, are reserved and recommended for congenital prolapse as seen in virgins and nulliparas because of abnormal weak structures.

(3) A round ligament suspension and utero-sacral plication with suitable plastic operations are best for women in the child-bearing period notwithstanding frequent recurrence.

(4) A well performed perineorrhaphy is the most important link in the chain of repair and should be supplemented by any necessary steps in order to preserve proper function of the pelvic floor.

REFERENCES

1. Goff, B. H.: An Evaluation of the Bissell Operation for Uterine Prolapse. Surg., Gyn. & Obst., **57**:763-771 (Dec.) 1933.
2. Counselor, V. S.; and Stacy, L. J.: Results of Various Operations for Prolapse of the Uterus. Collected Papers of the Mayo Clinic, **22**:331-334, 1930.
3. Everett, H. S.: End Results with the Watkins Interposition Operation. Surg., Gyn. & Obst., **61**:403-405 (Sept.) 1935.
4. Baer, J. L.; and Reis, R. A.: The Interposition Operation for Prolapse of Uterus: A Statistical Study of 91 Consecutive Operations and Analysis of the End Results. Amer. Jour. Obst. & Gyn., **17**:233-239 (Feb.) 1929.
5. Coventry, W. A.; and Moe, R. J.: Prolapse of the Uterus. Amer. Jour. Obst. & Gyn., **25**:257-261 (Feb.) 1933.
6. Cattell, R. B.; and Swinton, N. W.: Interposition of the Uterus in the Treatment of Cystocele: The Watkins-Wertheim Operation. Surg. Clinics N. A., **15**:1645-1652 (Dec.) 1935.
7. Shaw, W. F.: The Treatment of Prolapse Uteri with Special Reference to the Manchester Operation of Colporrhaphy. Amer. Jour. Obst. & Gyn., **26**:667-686 (Nov.) 1933.

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DISCUSSION (*Abstract*)

Dr. Guy L. Hunner, Baltimore, Md.—I think we all agree with Dr. Cofer in the general principles enunciated for the selection of those patients who should be operated upon by the vaginal approach. Most of us also agree that too many women with uncomplicated procidentia are still being subjected to the more hazardous and more depleting abdominal operation. As a rule, it is best for the individual patient to have that operation which the surgeon in charge can do best, but it is unfortunate for these procidentia cases as a class that many surgeons without special training in pelvic work are still accepting them for the more dangerous and less effective abdominal operation.

After enumerating the various conditions which require special consideration, such as enterocele and prolapse in virgins and nullipara, preservation of the child-bearing function, and the presence of tumors and inflammatory processes, Dr. Cofer has properly limited the discussion to the best methods of treating procidentia uteri by the vaginal route. Shall this be by one of the colpocleisis methods similar to Le Fort's operation, by an exaggerated general repair as in the Donald-Fothergill or Manchester operation, by one of the interposition or transposition methods like the Wertheim-Watkins operation, or by vaginal hysterectomy?

I always have a feeling of futility in entering such a discussion. The conditions vary so greatly in different patients that one soon learns that there is no standard best method of approach. We plan an operation for the individual patient and finish with an entirely different procedure. It is only experience that teaches us how best to cut our cloth, and to sew it together again, in a manner to give the patient the best result. One or two points learned in a fairly limited experience may help some of those just entering in this work.

It is well for the beginner to remember that the bladder, the rectum, and the pouch of Douglas form three potential hydraulic bags, and that an hydraulic bag is a powerful ram for the slow, insinuating separation of parts. No matter how carefully we coapt the various uterine ligaments to form our inner line of defense, and how skillfully we build up our second line of defense with the levators and the muscles and fascia forming the perineal body, we are inviting future trouble if we have not given our chief attention to the elimination of the dynamic effects of the three potential hydraulic bags.

In closing the interposition operation the vesicouterine segment of the peritoneum should carefully be isolated and attached by two or three plain catgut sutures in a transverse line across the upper posterior face of the uterus. Then when we close the anterior colporrhaphy incision across the anterior face of the uterus the first two interrupted No. 2 chromic catgut sutures should carefully include the strong musculo-fascial tissues immediately beneath the symphysis pubis. Henceforth the bladder does its distending within the abdominal cavity and not downward through the vestibule region.

If rectocele has been a feature of the prolapsus I like a modification of the operation of the late A. B. Spalding, of San Francisco (*J.A.M.A.*, 79:706, 1922). Carry the large oval denudation of the posterior vaginal wall well up against the posterior cervix so as to have easy access to the uterosacral ligaments. Before closing the posterior colporrhaphy incision with interrupted chromic catgut sutures do away with the rectal pouch by passing a fine silk or linen suture, first through the left uterosacral, then through the muscular coat and into the submucosa of the anterior left rectal wall just above the internal sphincter, go across the rectum and pick up the muscles and submucosa on the right wall and finish by carrying the suture through the right uterosacral ligament. Before tying this a second suture is passed in like manner, beginning slightly farther back on the tissues of the uterosacral and crossing the lower rectum at a plane slightly higher than the first suture. These two buried permanent sutures are then tied, the one last placed being tied first. We have thus done away with the air bag which otherwise tends to work through the posterior colporrhaphy repair.

Both Moschowitz (*Surg., Gyn. & Obst.*, 15:7, 1912) and Ward (*J.A.M.A.*, 79:709, 1922) have emphasized the frequency (previously unrecognized) of enterocele, and Ward shows how easily the hernia of Douglas' sac is recognized if we but keep it in mind, and how to extirpate the sac either when it occurs alone or in association with rectocele. If enterocele occurs with rectocele the pouch of Douglas should be excised before doing the high fixation of the rectum. If hysterectomy is indicated the uterus is first removed, thus simplifying the exposure and removal of the enterocele sac and the identification of the uterosacrals, to which the rectocele sac (if present) is fixed before approximation of the uterosacrals.

In the vaginal hysterectomy operation I find it helpful when tying and cutting across each of the major ligaments of the uterus to leave the ends of the ligature material long, and, for identification, to place a clamp on the end of each. Then in closing the peritoneum with a purse string of plain catgut the round ligaments, the broad ligaments (cardinal ligaments), and the uterosacrals are incorporated. Finally, with a chromic catgut all the ligaments are brought together with a second reinforcing purse string, and the long ends of this are used to drag forward the focal point until its tissues are caught in one or two of the first interrupted chromic sutures closing the anterior colporrhaphy wound just beneath the symphysis. This lifts the bladder to a high position.

In the past two years I have had two cases of procidentia accompanied by very unusual conditions.

A patient with a profound mental depression had spent several months in a psychiatric hospital. Pelvic examination revealed a mass protruding about 10 centimeters beyond the vulva and composed of thickened vaginal walls. At the apex of this mass the greatly enlarged cervix showed multiple retention cysts, many of these showing gray and yellow contents. The portiovaginalis of the cervix was much elongated, and the fundus, slightly larger than normal, apparently was being held fairly high in the pelvis by its ligaments.

A month after the correction of this physical deformity by a vaginal hysterectomy the patient read the newspaper and showed a lively interest in details of her home life. A year later she seemed entirely normal mentally, optimistic and happy.

The second patient received her injuries during a forceps delivery of her first child. In addition to a wide separation of the symphysis pubis, and complete procidentia of the genitalia and bladder, this patient had a large vesicovaginal fistula and complete destruction of the urethr[illegible]. The impossibility of restoring a functioning uret[illegible]r made it imperative that we provide some method of caring for the patient's incontinence, which represented the most serious aspect of her deformity. She weighed 267 pounds, having lost 91 pounds by dieting for the previous six months. The obesity made the prospects of a uretero-sigmoid anastomosis by the Coffey method an unattractive procedure, so I decided on vaginal removal of the prolapsed pelvic organs, vagina and bladder, and implantation of the ureters into the anterior rectal wall by the vaginal route. This was done on May 16, 1935, and at my request the patient returned for observation on February 27, 1936, when we found that both ureters were functioning actively and urine gathered through the rectal speculum from each ureter showed only an occasional leukocyte. The two-hour intravenous 'phthalein test, the urine being collected through the anus, showed: first hour, 300 c. c., 40 per cent; second hour, 175 c. c., 15 per cent.

Dr. John W. Turner, Atlanta, Ga.—Prolapse always indicates that the uterosacral ligaments and the cardinal ligaments in the bases of the broad ligaments have been torn or stretched. The associated rectocele and cystocele indicate injury to the pelvic floor.

The injuries permitting prolapse to develop occur during labor. What particular feature associated with childbearing can produce an injury or overstretching of the uterosacral, so-called cardinal, and uterovesical ligaments? During normal labor the longitudinal uterine fibers pull the lower segment upward and push the child downward and so the head passes through the cervical canal which keeps its relative level in the pelvis. Even a large child does not cause overstretching of these ligaments. Passing between their medial ends, the tendency would be to push these ends outward and to shorten rather than overstretch the uterosacral and cardinal ligaments. There is a pernicious

habit which is fortunately becoming more and more infrequent, namely, the practice of having a patient use her abdominal muscles during labor. In spite of the knowledge that no possible good can be secured by permitting a patient to use the abdominal muscles before complete dilatation of the cervix has been obtained, the attendant nurses constantly exhort the patient to "bear down." The entire force of the abdominal muscles tends to drive the uterus downward. The round ligaments afford only a stabilizing and balancing effect. The uterosacral and cardinal ligaments are the only structures holding the uterus in its place and affording resistance to the downward push of the abdominal muscles. It is no marvel that they are torn or overstretched. It would be interesting to question all patients suffering from prolapse of the uterus in regard to the birth weight of their babies and in regard to their "bearing down." It is probable that all cases would have exerted their abdominal muscles during the first stage and that the baby's birth weight has not played a prominent part in the production of the prolapse.

The bony pelvic ring offers greater resistance to a large head preventing descent of the uterus and head in these cases, while the uterus carrying a small child would meet less resistance at the pelvic brim and so would be pushed sufficiently low to cause tearing of the uterosacral and cardinal ligaments.

The problems of repair of prolapse are the same whether the patient is in the childbearing age or has passed the menopause. In the first group less liberty in the use of the available material is permitted. The childbearing function must be preserved. Here we may use the advancement of the bladder with high suture of the uterovesical ligament and anterior colporrhaphy with excision of redundant vaginal mucosa combined with shortening of cardinal ligaments and repair of perineum. A cervix infected or otherwise diseased should be amputated or treated by cauterization. It should be remembered, however, as Dr. Cofer has stated, that a high amputation of the cervix virtually prohibits childbearing. In the second group the interposition of the fundus of the uterus below the bladder, combined with the procedures mentioned above which may be used when the function of pregnancy is to be preserved, will give good results. If there are indications for the removal of the uterus, the cul-de-sac should be obliterated by the Ward method of suturing the uterosacral ligaments together. The suture of the uterosacral ligaments in the method advocated by Ward is a distinct and valuable contribution to the technic of repair in prolapse.

Dr. Cofer (closing).—Fifty-two cases out of 96 in a ten-year period are reported as having been critically examined by me, and in none was a complete failure found, though it was necessary to do the recommended vaginal hysterectomy in one case in which a Watkins interposition without the fixation stitch had been done. In the interposition operation, there were two partial anatomical failures and four partial symptomatic failures and strange to say, the most pronounced anatomical failure, a prolapse of the fundus, grade two, had produced no symptoms though operation had been done six years prior to the examination. A persistent cystitis seems to be the predominating symptom and a prolapsing fundus is the weak point anatomically. However, I feel that proper selection of cases and careful anchoring of the fundus will almost entirely eliminate both symptomatic and anatomic failures in this operation.

The vaginal vault prolapse with a shortened vagina is the weak point in the modified Mayo vaginal hysterectomy which can be largely, if not entirely, corrected by careful technic and the use of the Ward method of cul-de-sac closure.

There will always be a few failures in these operations, as well as in any surgical procedure, but the advantages so far overbalance any possible disadvantages that, in my opinion, no careful gynecologist would deny their usefulness.

ROENTGEN RAY THERAPY IN FIBROUS ANKYLOSIS OF THE JAW*

By A. R. MacKenzie, M.D.
Huntington, West Virginia

In 1923 I treated successfully my first case of fibrous ankylosis of the jaw with roentgen ray therapy. My findings were reported to our local medical society, the Cabell County Medical Society of Huntington, West Virginia, the following year. During the period, from 1923 to 1936, I have had 29 cases which have been treated with roentgen ray therapy, with only one partial failure. Since 1923 I have made a diligent search of the literature, but so far have found no reference to the use of this agent in the treatment of fibrous ankylosis of the jaw. As to sex, my patients have been about equally divided, 16 males and 13 females. Eight of this series were bilateral, and 21 were unilateral. In all of those having bilateral involvement there was a history of pre-existing infection in the tonsils with extensive soft part infection. In those having unilateral involvement there was a history of a pre-existing infection from carious teeth with surgical interference previous to x-ray therapy, and in nearly all of these cases a nerve block was used. Before coming under my observation, the period of duration was from 3 months to 3 years. Ages ranged from 9 years to 34. I must admit that I have carefully selected those which I have treated and within the scope of this paper are included only those of the articular type, fibrous and non-bony in nature. Nor has there been included any case of scarring of the cheek or myositis. In one case of this series there was a pre-existing Albers-Schonders disease (marble bones) which the patient did not realize existed and which was only picked up

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in general routine examination and had been of no inconvenience to the patient until a carious tooth was extracted with a unilateral ankylosis on the left side, which, in my opinion, was only a coincidence. In all of these cases a careful radiographic examination was made and there was apparently no bone involvement. The condylar surface and the glenoid cavity were found to be smooth and regular, and there existed no deformity of the neck of the condyle. There was one case of an old multiple fracture with painful unilateral involvement. In this series surgical intervention was used in five cases with only partial, or no definite, improvement. I must admit, however, that the surgery in these cases was not always of the highest order. It is not my purpose to burden you with the anatomical structure of the temporo-mandibular joint.

In the extra-articular type the condition is usually due to injuries to the coronoid process or to myositis. The latter may occur from a number of causes: (1) abscess due to osteomyelitis or dental infection, (2) gumma due to syphilis, (3) presence of an infected impacted third molar, or (4) any condition which interferes with the muscles of mastication and tends to produce fibrosis upon healing. The following types of infections may be considered as causative factors: pyemia following septicemia, otitis media, osteomyelitis, periostitis, and in some cases gonorrhea and metastatic malignancy.

In the specific etiology of this series the causative factor appears to have been definitely infectious, either from an infected throat with extension, or from infected teeth, aggravated by traumatism (either surgical or from movements of the joint in which considerable pressure was exerted upon the structures of the joint in an effort to masticate solid food in the presence of an inflammatory process).

The only partial failure that I have experienced was a case, occurring two years after surgical intervention, of osteomyelitis of the lower maxilla with no bony involvement of the joint, but associated with an apparent fibrous ankylosis. While the result has not been entirely satisfactory to date, it is too early for final report. Although the patient has definitely improved with 50 per cent restoration of function, allowing him to protrude his tongue and masticate his food, and to take sufficient nourishment for his body maintenance, I am not yet entirely satisfied with the end result because he has not had a complete restoration of function, nor has he been able to open his mouth completely. This has been more or less of a disappointment to me because of the results I have obtained in the other cases.

In every case I have endeavored to rule out bony ankylosis and myositis, which I am free to admit are at times very difficult to differentiate, even for a skilled diagnostician. In all of these cases there was apparently a normal symmetry of both sides of the face and little or no pain elicited by examination or movement. A certain amount of imaginary pain probably was induced by the development of a mild psychosis with an inferiority complex, which was pronounced in a number of individuals after a certain length of time, due to their inability to open their mouths.

I am not bringing to you this method as a definite one of choice, nor to the exclusion of a surgical method which will in many cases be necessary. But I am drawing to your attention a useful method, which, in my opinion, should be tried, especially when in doubt of the diagnosis, before any surgical method is adopted. It cannot be definitely proven in all my cases that we were dealing with a fibrous ankylosis. I have proof for only 5 cases in this series where there was found at operation a predominance of fibrous tissue in and about the joint in amounts far above that normally found. However, it seems reasonable to assume that the same conditions existed in the other patients, since the same history, symptoms and signs were presented. Even in cases of less than three months duration, the question in my mind is, "what would have been the ultimate result if they had not been treated?" It has been our policy in the treatment of those cases where symptoms had existed at least three months and in which a bony ankylosis or myositis can be excluded, to give our patient the benefit of the doubt with immediate roentgen ray therapy, because we have everything to gain and nothing to lose, and no permanent damage can be done. If for no other reason than the cosmetic effect and the possible complete normal restoration of function, this simple method should at least be tried. Moreover, surgical intervention, from reports, is not always entirely satisfactory.

In conclusion, may I hold your attention a few moments longer to cite brief details of the most striking case in this series, and the first reported.

Mr. G. B., age 32, a white man, gave a history of no importance except that he had had a chronic throat infection for 9 years before he came under my obser-

vation. He had had peritonsillar abscesses evacuted and drainage established 19 times, because he refused to have his tonsils removed. Three months after his last drainage for a peritonsillar abscess the patient noticed that he could no longer open his mouth sufficiently to introduce the handle of a spoon so that it would pass between his teeth. His condition became progressively worse, and he developed an acute arthritis. He was then referred to me by a local orthopedic surgeon for general radiographic examination, which revealed arthritis of a knee, ankle, elbow, wrist, and cervical spine. He held his neck rigid and was taking ¼ to ½ grain of morphine every 3 hours, with little or no relief from his pain. He had developed an inferiority complex to such an extent that he would no longer eat at the same table with his family. The patient had been subjected to every known mechanical and surgical method, by practically every surgeon in the city, which included forcible attempts to insert chisels with leverage between the teeth under deep anesthesia. The last operation performed on him consisted of cutting down on the joints, resecting the scar tissue in and about the joint, with an accidental nicking of Stenson's duct on the left side, resulting in a fistulous opening to the outer cheek with marked excoriation of the tissues. The joint was so thoroughly dissected loose that he was told by the surgeon, when the dressings were removed, "to keep his chin up so that it would not drop." But having his bandages removed for the last time, he found, much to his surprise, that he had complete occlusion, his jaws being solidly locked so as to prevent the insertion of even a razor blade between the teeth. Six months after the onset, in hysterical desperation, after this last masterful piece of surgery, in a fit of maniacal excitement he knocked out two of his front teeth with a hammer and nail so that he could use a glass tube to suck thin soups and milk for his existence. In this condition he lived for two and one-half years before coming to see me.

Because of this man's pitiful condition, I was moved to practice empirical medicine, against my better judgment. I felt that if we could cut down with roentgen ray treatment the local infection in his tonsillar tissue, we could probably relieve his general arthritis. This was doubtless a far-fetched hit-or-miss method, of which I must confess I was more or less ashamed. But it was a revelation to me, when 3 weeks after one ordinary so-called subintensive dose of roentgen ray he walked into my office with his mouth half open. This was a great relief to the individual after three years of suffering.

I returned him then to a nose and throat man for removal of his tonsils. The nose and throat specialist, who was bitterly opposed to roentgen ray therapy in any form, told the patient after removing his tonsils that he certainly hoped he would never get another case which had had x-ray therapy on the tonsils because he had never seen so much scar tissue in an individual's throat, which he said was produced by the x-ray, overlooking the fact that for each time an abscess was opened scar tissue formation followed. After the removal of his tonsils, two more doses of x-ray were given. Following this, the infection completely cleared up and after 13 years he has had no recurrence. He has gained in weight, has had no return of joint symptoms, and is apparently again in normal health.

Technic.—Formerly the treatment given consisted of 140 kv., 5 ma., 5 mm. aluminum filter, suitable cone to cover field, 16 cm. distance, 15-20 minutes. This was practically equivalent to 350 R.

Later, I employed 200 kv., 0.25 mm., copper filter, 50 cm. distance, suitable cone, 350 roentgens, average number of treatments given 2-5, time interval from ten days to two weeks.

The results were almost the same, and in my opinion there was no material difference between the two technics.

SUMMARY

(1) Roentgen ray can be used successfully in the treatment of selected cases of fibrous ankylosis of the jaw.

(2) The use of roentgen ray therapy in fibrous ankylosis of the jaw is not herewith advocated as a substitute for surgical intervention, but is recommended as a method of primary choice for the following reasons: (a) scar tissue formation resulting from surgical operation, especially marked in certain individuals, and (b) the successful results obtained in the treatment of this series of 29 cases.

(3) The results obtained in the treatment of fibrous ankylosis of the jaw are equally comparable to and as successful as those obtained in selected cases of fibroids of the uterus, and also keloids.

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DISCUSSION (*Abstract*)

Dr. Max Kahn, Baltimore, Md.—Dr. MacKenzie's results show that fibrous ankylosis of the jaw which heretofore has largely been treated by heat, manipulation, massage and by surgical intervention, will in many cases yield to irradiation. My experience in treating painful joints with irradiation is also encouraging. The patients referred for x-ray therapy had painful joints due either to subdeltoid bursitis, arthritis, fractures in or about the joints, or due to some undetermined cause frequently called neuritis. Clinically and symptomatically the majority of the patients improve, the pain is lessened or completely disappears and they are better able to move the joint. The underlying pathologic condition in the majority of the cases treated remains, but in the subdeltoid bursitis there may be absorption of the bursa. I should like to report briefly a case of a very painful shoulder joint following a comminuted fracture of the outer third of the clavicle. The patient was a woman over 70 years of age. Six weeks following the fracture there was displacement of a large fragment at right angle, with bony union. Surgical removal of the fragment was considered inadvisable, although pain prevented the patient from

sleeping. She was treated by deep irradiation once a week for 6 weeks, receiving 20 r at a treatment, and her pain completely disappeared. It is now over three months and there has been no recurrence of pain.

I feel that irradiation is a useful measure in many cases of painful joints, either with or without ankylosis, and should be used more frequently in conjunction with other forms of treatment or alone as the case may require.

DRAINAGE AS A FACTOR IN THE TREATMENT OF LOBAR PNEUMONIA*

By CONLEY H. SANFORD, M.D., F.A.C.P.
Memphis, Tennessee

That the treatment of lobar pneumonia is one of the unsolved medical problems is attested to by the fact that the mortality rate has not been materially reduced. Yandell Henderson and his co-authors,[1] in commenting on the subject, state that the organisms involved in lobar pneumonia are as well known as those of typhoid fever and diphtheria; that the same methods of bacteriology and serology have been applied by as able investigators, but that while the mortality rate of such diseases as typhoid and diphtheria has declined to a very small figure that of lobar pneumonia has remained essentially unchanged. They believe that there are factors involved in pneumonia which are not applicable in other infectious diseases; that those factors are occlusion of an infected organ and lack of drainage.

Recent advances in our knowledge of the pathogenesis of lobar pneumonia and the physiology of the respiratory tract enable us to comprehend the rationale of drainage as an effective method of treatment of that disease.

The work of Pruden and Northrup in 1889, of Silfast in 1899, of Hildebrandt, Rosenthal, Barthel and others has been quoted by Coryllos and Birnbaum[2] and Wadsworth.[3] They established the fact that the mucosa of the lower portion of the trachea, the bronchi, the bronchioles and the alveoli is normally sterile. Sterility is maintained by a number of factors as follows: (1) the action of the ciliated epithelium; (2) bactericidal qualities of mucus; (3) phagocytosis; (4) the cough reflex; (5) respiratory movement; and (6) peristaltic action of the air passages. Henderson[4] found that peristaltic waves, beginning in the alveoli and progressing rhythmically toward the trachea, occur in the normal man at the rate of one wave every seven or eight minutes. As a result of the combined action of these factors it has been said that the lungs normally are the best drained organs of the body.

In view of the above if we concede that the organisms causing lobar pneumonia gain entrance to the lungs through the air passages then it must follow that infection is preceded by something which lowers the resistance of the respiratory tract against invasion. As a matter of fact in order to produce pneumonia experimentally in a previously healthy dog it is usually necessary to depress respiratory movement generally accomplished by administering large doses of barbiturates prior to insufflation of the infectious agent.

Blake and Cecil[5] have proven conclusively that the portal of entry of the pneumococcus in producing lobar pneumonia is the air passages. By injecting virulent pneumococci into the trachea of monkeys they were able to produce the disease which was analogous in all essential features to that in man. They found also that after intratracheal injections septicemia occasionally occurred in monkeys which never developed pneumonia, indicating that septicemia may occur from invasion of the blood stream in the walls of the trachea or bronchi. They found, further, that after injecting virulent pneumococci subcutaneously and intravenously septicemia developed, but not in a single instance did pneumonia result.

It was also demonstrated by Blake and Cecil[6] that the disease begins at the hilus and extends progressively toward the periphery of the lobe. When the disease is in the stage of engorgement at the hilus, the periphery is normal; red hepatization near the hilus is accompanied by engorgement at the periphery; gray hepatization near the hilus, red hepatization at the periphery and when resolution occurs at the hilus, the periphery is still in the stage of gray hepatization. It was their opinion that the infection spread from the hilus by way of the interstitial framework and the lymphatic system.

There are many facts which indicate that lobar pneumonia is primarily a pneumococcic lobar atelectasis. It has already been said that infection reaches the lung by way of the air passages and that the disease begins at the hilus and extends progressively toward the periphery. I

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should like to discuss the remaining evidences under the following headings: (1) the specificity of the etiologic organism; (2) the physical signs of early lobar pneumonia; (3) the size of the involved lobe; (4) roentgenologic evidence; and (5) miscellaneous.

Does it not seem more than mere coincidence that in essentially all cases of lobar pneumonia the pneumococcus is the etiologic agent, while bronchopneumonia is produced by a multiplicity of organisms? The pneumococcus produces a very thick, tenacious exudate, rich in fibrin, which is capable of plugging a large bronchus, especially since the lumen of the bronchus has already been narrowed by inflammatory infiltration and edema. On the other hand, the streptococcus and other organisms commonly responsible for bronchopneumonia cause a thin type of exudate which may be capable of blocking bronchioles and smaller bronchi, but is rarely capable of occluding a large bronchus.

The physical signs of early lobar pneumonia are essentially the same as those of atelectasis. In the first twenty-four hours or so of lobar pneumonia we usually observe increased respiratory rate, limitation of motion on the affected side, decreased tactile fremitus, dullness on percussion and diminished breath sounds over the involved lobe. Indeed, in many instances these findings persist throughout the course of the disease. It is only later that we are able to elicit tubular breathing, which we are prone to call the characteristic auscultatory finding of lobar pneumonia.

Contrary to the former teachings of pathologists, the involved lobe in lobar pneumonia is smaller than normal. When the thorax is opened at necropsy, the normal, air-containing lung tissue collapses, but the consolidated, pneumonic lobe retains its premortem size. By camparison, therefore, this gives the impression that the diseased lobe is larger than normal. If, however, prior to opening the thorax the trachea be ligated to prevent collapse of the normal lung tissue, it will be seen that the involved lobe is actually smaller than normal. This has been demonstrated by Coryllos and Birnbaum,[2] also by Van Allen and Wu,[7] the latter ascribing the smaller size of the pneumonic lobe to an increase in elastic tension.

Roentgenologic study indicates not only that the involved lobe is smaller than normal, but also shows a striking similarity between lobar pneumonia and lobar atelectasis in that, contrary to previous belief, the diaphragm is elevated on the affected side or the mediastinal structures are displaced toward the affected side, or both. It seems that in children, in whom the mediastinal structures are labile, the heart and trachea are displaced toward the involved side, whereas in adults, in whom the mediastinum is more or less fixed, the decrease in lung volume is compensated for by elevation of the diaphragm on the affected side. Wu[8] found in 40 consecutive cases of lobar pneumonia, mostly adults, that five showed displacement of the heart and trachea toward the affected side and in 22 the diaphragm was elevated on the affected side. McNeil, MacGregor and Alexander,[9] in discussing pneumonia in children, ascribed the displacement of the heart to traction exercised by shrinkage due to partial collapse of the affected lung, aided by push of the sound lung, which is frequently distended by compensatory emphysema. Graeser, Wu and Robertson[10] reported that 18 of 29 cases of lobar pneumonia in which the diaphragm could be visualized showed an elevation on the affected side and that in the other 11 cases the diaphragm was obscured by fluid or dense consolidation so that it could not be stated whether or not there was elevation later in the disease. I have failed to find an authentic report of depression of the diaphragm on the affected side or displacement of the heart and trachea away from the affected side in uncomplicated lobar pneumonia.

Coryllos and Birnbaum[2] enumerate the following additional indications that lobar pneumonia is primarily a pneumococcic lobar atelectasis: (1) the lobar distribution of lobar pneumonia; (2) the more frequent localization in the inferior lobes, especially the right; (3) the sudden onset of the disease; (4) the relationship between "colds" and the development of pneumonia; (5) the atypical, abortive and delayed forms; (6) the crisis, which cannot be explained by immunologic factors alone.

Regarding the treatment of lobar pneumonia, it makes no material difference whether or not we subscribe to the theory that the disease is primarily a pneumococcic atelectasis; the fact remains that pneumonia is an infection of lung tissue. When there is a localized area of tissue infected, regardless of whether it is bone, subcutaneous tissue or lung tissue, the indication for treatment is drainage. When a natural route of drainage is available, it seems logical that the natural route should be selected. Nature has

made adequate provision for drainage of the respiratory tract, as previously pointed out.

It is a well known fact that the usual treatment of lobar pneumonia, in my own locality at least, combats nature's own efforts to promote drainage. As a rule the patient is permitted to assume the position in bed that he prefers, which is usually with the affected side downward; he is given opiates freely, which depress respiration and cough, nature's most effective measures for drainage; the chest is encircled with mustard plasters, antiphlogistine, pneumonia jackets, which further embarrass respiration.

The method of treatment advocated was first called to my attention in connection with postoperative pneumonia. It is now fairly generally conceded that postoperative pneumonia is primarily atelectasis: that during prolonged and profound anesthesia mucus is inspired, occluding a bronchus. If this occluding substance, which contains pathogenic organisms, is allowed to remain in the bronchus the functionless, atelectatic lobe becomes infected and postoperative pneumonia ensues. It has been shown by Yandell Henderson[11] and others that if carbon dioxide in oxygen is administered at the end of anesthesia, the incidence of postoperative pneumonia is markedly reduced.

Furthermore, it has been the experience of many of us that a most effective method of treating postoperative pneumonia consists of withholding, in so far as possible, opiates and other respiratory depressants; keeping the patient on the unaffected side, thereby soliciting the aid of gravity in promoting drainage; and the administration of carbon dioxide in oxygen by inhalation at frequent intervals for the purpose of stimulating respiration and cough. Using this method of treatment it is amazing how frequently the fever and all other symptoms subside within a few hours.

In advocating this method of treatment for lobar pneumonia it would seem, perhaps, that one must assume the correctness of the theory of atelectasis as the initial factor in the pathogenesis. This is not entirely true, however, since a large part of the inflammatory process in pneumonia involves the lining of the bronchi, bronchioles and alveoli—discharging its exudate into the air passages themselves—and it seems plausible that the removal of this excessive amount of infectious material would lessen the extent of invasion of interstitial tissue. It is admitted that, as pointed out by Blake and Cecil,[6] the disease spreads partly through the lymph spaces in the interstitial tissue of the lung from the hilus to the periphery and complete, immediate drainage by way of the air passages of this interstitial tissue is impossible. The same might be said, however, of an extensive and deep subcutaneous abscess. After incision, the surrounding inflamed and infected tissue does not drain completely and immediately, but it is more likely to drain eventually if it has a portal of exit than if it had none. Furthermore, the extension of pneumonia from the hilus to the periphery is probably not alone in the lymph spaces in the interstitial tissue, but also by direct extension along the lining of the air passages. It is hardly conceivable that the infection would spread from pharynx to trachea, thence to bronchus and at that point extend solely by way of the interstitial tissue.

The effect of this method of treatment is, obviously, more or less inversely proportionate to the duration of the disease at the time treatment is instituted. It has been my experience that pneumonia is frequently aborted when treatment is begun in the first twenty-four to forty-eight hours of the disease. The longer the disease exists, the more extensive pulmonary cellulitis becomes and, incidentally, the more likely septicemia is to develop.

If pneumococcic septicemia complicates pneumonia, the only argument for drainage as a method of treatment has been well expressed by Scott[12] in discussing septicemia in general. He said:

> "The most important principle in the treatment of septicemia is the eradication or exclusion from the circulation of all foci that are reinfecting the blood."

A discussion of drainage in the treatment of lobar pneumonia would not be complete without a few words about bronchoscopy. It is not my impression that bronchoscopic aspiration is necessary or desirable in all cases of lobar pneumonia. However, in those patients whose lungs fill up with an excessive amount of exudate in spite of all efforts, and who are on the verge of "drowning in their own secretions," there is no doubt that bronchoscopic aspiration, repeated every eight hours if necessary, is often life saving. I am thoroughly convinced that a few of my private patients who survived would have succumbed had it not been for that procedure. It should be emphasized that in the hands of a skillful bronchoscopist the operation can be performed in a very short time, with no shock and very little discomfort to the patient.

My favorable experience with this method of treatment in a small series of private patients made it seem desirable to try it out on a larger number. With that in view, the method was used exclusively on all adult cases of lobar pneumonia admitted to the John Gaston Hospital from April 15, 1935, to April 15, 1936. A more complete description of the technic of treatment and a detailed report of the year's experimental work will be published elsewhere. Briefly, however, 309 cases comprise the series. These cases were unselected; every adult case of lobar pneumonia admitted to the hospital was included. Of the 309 cases, 76 died, making a mortality rate of 24.6 per cent.

For purposes of comparison, the records of all adult cases of lobar pneumonia in the Memphis General Hospital, the predecessor of the John Gaston Hospital, for a period of ten years, from 1925 to 1934, inclusive, were reviewed. The lowest mortality rate of any year was 26.6 per cent; the highest, 36.6 per cent; the average, 31.3 per cent.

At first thought, it seems that the reduction in mortality rate was rather small. It is my impression, however, that the results are more real than apparent for the following reasons: (1) The work was carried out in a charity hospital; most of the patients were Negroes; all were underprivileged individuals; many suffered from syphilis, anemia, malnutrition and other complications. The above also applied in previous years, the records of which we are comparing with those of the experiment, but, I think, these factors would limit the degree of favorable results of any form of treatment for practically any disease selected for experimental study. (2) For the most part the wards were crowded, the nursing staff was inadequate and it was a physical impossibility for us to carry out the treatment as effectively as we should have liked. (3) The average duration of illness on admission, that is, before treatment was instituted, was four and one-half days. It has been stated above that the treatment, to be most effective, should be instituted in the early stages of the disease. (4) Forty and one-half per cent of the cases were infected with Types II and III pneumococci, indicating a rather virulent type of infection. (5) The percentage of septicemia was greater than for any of the previous years, 22 per cent. For the most part, this meant septicemia on admission, since blood cultures were obtained as soon as possible after the patient entered the hospital and the initial culture was positive in practically all cases with septicemia.

SUMMARY

(1) The physiology of the respiratory tract, as pertains to normal drainage, is reviewed.

(2) The pathogenesis of lobar pneumonia, with evidences pointing toward its being primarily a pneumococcic lobar atelectasis, is discussed.

(3) A method of treatment, designed to give drainage to infected lung tissue, consisting of keeping the patient on the unaffected side, withholding opiates and other respiratory depressants and administering carbon dioxide in oxygen for the purpose of stimulating respiration and cough, is advocated.

(4) A brief synopsis of the results of treatment of 309 cases by this method is given.

REFERENCES

1. Henderson, Y.; Haggard, H. W.; Coryllos, P. N.; and Birnbaum, G. L.: The Treatment of Pneumonia by the Inhalation of Carbon Dioxide. Arch. Int. Med., **45**:72 (Jan.) 1930.
2. Coryllos, P. N.; and Birnbaum, G. L.: Lobar Pneumonia Considered as Pneumococcic Lobar Atelectasis of the Lung: Bronchoscopic Investigation. Arch. Surg., **18**:190 (Jan.) 1929.
3. Wadsworth, A.: Experimental Studies on the Etiology of Acute Pneumonitis. Amer. Jour. Med. Sci., **127**:851 (May) 1904.
4. Henderson, Y.: The Physiology of Atelectasis. J.A.M.A., **93**:96 (July 13) 1929.
5. Blake, F. G.; and Cecil, R. L.: Production of Pneumococcus Lobar Pneumonia in Monkeys. Jour. Exper. Med., **31**:403 (April) 1920.
6. Blake, F. G.; and Cecil, R. L.: Pathology and Pathogenesis of Pneumococcus Lobar Pneumonia in Monkeys. Jour. Exper. Med., **31**:445 (April) 1920.
7. Van Allen, C. M.; and Wu, C.: Increased Elastic Tension of the Lung in Experimental Pneumonia. Jour. Clin. Inves., **11**:589, 1932.
8. Wu, C.: Visceral Displacement in Pneumonia: A Roentgenologic and Experimental Study. Radiology, **19**:215 (Oct.) 1932.
9. McNeil, C.; MacGregor, A. R.; and Alexander, W. A.: Pneumonia in Childhood. Arch. Dis. Child., **4**:170 (Aug.) 1929.
10. Graeser, J. B.; Wu, C.; and Robertson, O. H.: Physical Signs and Roentgenographic Findings in Lobar Pneumonia in Adults. Arch. Int. Med., **53**:249 (Feb.) 1934.
11. Henderson, Y.: Hyperventilation of the Lungs as a Prophylactic Measure for Pneumonia. J.A.M.A., **92**:434 (Feb. 9) 1929.
12. Scott, W. J. M.: The Principles of the Treatment of Septicemia. J.A.M.A., **105**:1246 (Oct. 19) 1935.

DISCUSSION (*Abstract*)

Dr. John Minor, Washington, D. C.—While it is my firm conviction that the further advance in the control of pneumonia will be along serologic lines; and further steps in this direction are being taken slowly but steadily, especially since the differentiation of many types of pneumococcus has become possible following the work of Cooper and others, any procedure which deals with the problem from a new angle and holds out hope of lowered mortality merits our serious attention. In the past ten years the two most discussed non-specific measures have been oxygen therapy and artificial pneumothorax. The results obtained by their use go far to strengthen the opinion that the hope of the future lies in specific therapy; that while such methods have

their place, they cannot, from the evidence so far adduced in considerable series of cases, be shown materially to modify the mortality from pneumonia.

The question of pathogenesis is an important one, and though it is true that Blake and Cecil showed that in monkeys the disease usually began at the hilus, spreading thence to the periphery, it has long been believed, and recently been convincingly shown by Graeser, Wu and Robertson, that the process begins sometimes at the hilus, sometimes at the periphery, and occasionally in between. The theory of lobar atelectasis as the precipitating cause in lobar pneumonia has failed to receive confirmation since the early work of Coryllos, Henderson *et al.*, and recently Robertson, Coggeshall and Terrell brought out evidence strongly contradicting this theory and finding no evidence of bronchial plugging with atelectasis, in experimental pneumonia; and it is interesting to note that though an early report from the New Haven group gave a mortality of 7 per cent in one series treated on the theory of relief of atelectasis and hyperventilation, no later similar reports have come from this clinic.

It would seem difficult to accept the concept of lobar pneumonia as a cellulitis of the lung, or to compare it with a localized abscess or pus collection elsewhere in the body. Pathologically, the striking features of lobar pneumonia are the presence of large amounts of fibrin in the exudate, and the fact that the alveolar and bronchiolar walls are rarely importantly involved and rapidly return to an entirely normal state after resolution is complete. As a pathologist says: "While it must be admitted that the pneumococcus is capable of producing a suppurative and necrosing process (in lobar pneumonia) it is probable that this is a rare event."

With regard to the comparison between lobar and bronchopneumonia, it must be remembered that Type III pneumococcus is a much more frequent cause of bronchopneumonia than either Type I or Type II, though the secretion it produces is notably the most viscid and tenacious produced by any type of pneumococcus.

While it is doubtless true that the presence of mucous plugs in bronchi does produce a certain amount of atelectasis in lobar pneumonia, and their removal has been known to lessen dyspnea in some cases, it seems to me very doubtful that the element of drainage can play any part in recovery from lobar pneumonia: the fibrinous secretion characteristic of this infection is not capable of drainage: it is dry and rather solid; and unchanged physical signs with roentgen ray evidence of continuing consolidation persist regularly from one to three days following a crisis: the important absorption takes place through the lymphatics.

Postoperative pneumonia represents commonly, not one, but one of several conditions, for example, massive collapse, bronchopneumonia from the aspiration of infected material, embolic pneumonia, infarction, or the flare-up of a pre-existing bronchial or mucous membrane infection. It is probable that a considerable proportion of those which clear up rapidly fall in the group of atelectases, and these, as we all know, have fairly definite physical signs and x-ray appearances and respond remarkably to measures directed to the relief of atelectasis. The other groups, it seems to me, run the course which one would expect from their etiology and pathology. Donald King showed in a controlled series at the Massachusetts General Hospital no reduction in mortality or morbidity in cases treated postoperatively with carbon dioxide inhalations.

Van Allen and Wu explained the elevation of the diaphragm on the affected side by an increase in the elasticity of the lung due to dilated capillaries and interstitial inflammatory fluids and cells; atelectasis is not necessary to explain the relatively slight shifts which occur.

I should like to ask Dr. Sanford whether in the treatment of these patients he used a tent or inhalators; also, on what indications and in what dosage opiates were used, if at all. I must say that I am in entire agreement as to the general tendency to the overuse of opiates in pneumonia: the doses are likely to be too large, too frequently repeated, and used in too routine a way. It is surprising how much can be accomplished in relieving without narcotizing the patient using doses of morphia as small as 1/12 or 1/16 of a grain, repeated as judgment indicates: except for the relief of pain, chest swathes and adhesive strapping are in my experience not helpful.

In conclusion, I would say that I sincerely admire Dr. Sanford's effort to apply and evaluate a new method in the treatment of lobar pneumonia, but that I find it difficult to give an equal importance with him to the role of atelectasis; that there seems to me to be strong evidence both against this theory and against the effectiveness of hyperventilation and bronchoscopy, except in carefully selected cases. It would be interesting and worth while to run a considerable series treated, when possible, within the first forty-eight hours of the disease, with suitable controls not so treated. It seems to me, while no possible adjuvant should be neglected, no path of possible aid left unexplored, that the endeavor most likely to result in real diminution in the still dreadful mortality from lobar pneumonia is the organization of methods and means in many localities for the obtaining of serum, the typing of all pneumonias, and the training of the practitioner in the technic and indications for the use of serum. We are still a long way from success, but there are truly encouraging signs and specific therapy on the basis of humoral immunity seems to me the hope of the future.

Dr. Henry M. Thomas, Jr., Baltimore, Md.—The paper which has just been presented by Dr. Sanford raises extremely interesting and important considerations in regard to the treatment of lobar pneumonia. The figures which Dr. Sanford gives are really very impressive. Under his form of drainage therapy, the mortality for a large number of cases was slightly lower than the lowest figures during the previous ten years in the same hospital. As the author himself says, this is not conclusive but quite suggestive. We are all familiar with the tremendous variation in mortality figures of lobar pneumonia from year to year and from locality to locality. No lasting conclusions, therefore, can be drawn from any series which represents only a relatively short period of trial.

The drainage method is in no way competing with other methods of treating lobar pneumonia. The author does not ask us to give up serum therapy in appropriate cases, nor does he close the door for future advances in chemotherapy. On the other hand, Dr. Sanford is performing an extremely valuable service by pointing out the need for consideration of general measures which apply to the treatment of every case of lobar pneumonia, no matter how early or how late the patient may be

seen, no matter which type of organism is responsible, whether the patient is treated at home or on the medical service of a hospital especially fitted for all the complicated requirements of modern serum therapy.

I had an interesting experience as "pneumonia specialist" in the Army during the Great War. This meant that after a few months of training at the Rockefeller Institute I was entrusted with all the serum treatment of Type I pneumonia cases at Camp Meade. Our mortality in the first fifty cases so treated was 6 per cent; and of the three cases who died, one had an enormous tuberculous cavity, one had been operated upon for empyema before the serum treatment was begun, and the third died several weeks later of streptococcus meningitis. If results such as these could be obtained in general practice in all types of pneumococcus lobar pneumonia, there would be no need for Dr. Sanford's work; but, on the other hand, it is only too clear that serum therapy is limited to specific types, must be administered early, and only by trained individuals. Even under these conditions, it is an extremely expensive form of therapy.

I am very much interested in Dr. Sanford's methods and results, and I shall watch with great interest the continuation of this work during the next few years.

Dr. Sanford (closing).—In answer to Dr. Minor's questions, carbon dioxide and oxygen gas were administered by inhalators exclusively. Opiates were used only in those cases in which rather severe pleuritic pain was present. All except about 3 or 4 per cent of our patients were reasonably comfortable without opiates. The usual dose, when given, was 1/8 of a grain of morphine and was repeated as infrequently as practicable, in several instances not repeated at all.

The purpose of this paper is not to belittle the use of serum or any other agent or method of treatment which offers some hope of reducing the mortality rate of lobar pneumonia. Its purpose, rather, is to present for your consideration a method of treatment which, to the author, seems logical and rational and which, in his experience, has proven to be of benefit.

CHRONIC CARBON MONOXIDE ANOXEMIA: CLINICAL SYNDROMES*

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The object of this paper is not to discuss the immediate effects of acute carbon monoxide poisoning as the incidence and the nature of this condition[1,2] are well known; but to consider the more remote effects of acute poisoning and the effects of chronic poisoning produced by frequent and prolonged periods of exposure to sublethal amounts, and to describe some of the characteristic syndromes.

The subject of chronic carbon monoxide poisoning has not received much attention in this country, although it has been freely discussed in foreign countries. Until recently a doubt prevailed as to the actual existence of chronic carbon monoxide poisoning. This was based on the assumption that long exposure to a low concentration was harmless. However, Sayers and Yant[3] proved conclusively that the symptoms and after-effects of a given blood saturation will be more severe if there has been a long exposure to a low concentration than if there has been a short exposure to a high concentration. This observation firmly established chronic carbon monoxide poisoning as a definite clinical entity.

In order to interpret properly the symptoms of chronic carbon monoxide poisoning, a brief review of the underlying pathologic changes as they occur in the blood and bodily tissues is essential.

The injurious effects of carbon monoxide are not directly due to the gas, which in itself is nontoxic, but to an anoxemia which is produced by replacement of the oxygen in the hemoglobin molecule by carbon monoxide for which it has a great affinity. Hence the term "anoxemia" would seem preferable to "poisoning." Unless the exposure is prolonged or frequently repeated, no morphological changes occur in the blood, the symptoms being confined to those of oxygen deprivation, and differ in no respect from anoxemia from any other cause. However, if exposure to the gas is frequent or prolonged, a relative polycythemia frequently develops with an increase in the hemoglobin content of the blood, probably the effect of a compensatory mechanism.

Gerbis[4] observed in dogs, and Williams and Smith[5] in rats daily exposed to sublethal amounts of carbon monoxide an increase in red cells and hemoglobin. A slight fragility of red cells occurred in rats, but only after gassing for more than 200 consecutive days, a condition which in rare instances leads to severe anemia. Either anemia or polycythemia was found to occur in soldiers suffering from chronic carbon monoxide anoxemia, as well as in civilians exposed to the gas in certain industrial pursuits. No diagnostically significant changes seem to occur in the white cells.

PATHOLOGIC ANATOMY

Organs whose functions depend on a rich blood supply such as the central nervous system and the heart muscle naturally suffer first from the effects of anoxemia; and the lesions produced are found most abundant in these structures, though all the organs may become involved. The nature and extent of the lesions depend largely

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upon the amount of gas inhaled, the frequency and duration of exposure, and to some extent upon the admixture of other noxious gases or vapors. The lesions are primarily vascular, and consist of venous and arterial congestion with perivascular infiltration and tendency to edema. Hemorrhages are common and occur in practically every organ. Nutritive disturbances of the vessel walls are followed by degenerative changes resulting in some instances in arterial spasm, thrombosis and gangrene. The hemorrhages vary in size from small petechiae to frank hemorrhages. Fatal cases of hemoptysis and epistaxis have been reported; and bleeding from the stomach, intestines and kidneys has been clinically observed; while hemorrhagic areas have been found in the myocardium, spleen and other organs at autopsy. The most damaging hemorrhages occur in the brain and meninges. There is a special predilection for the basal ganglia. As a result of these hemorrhages and perivascular infiltration, focal softening occurs with cystic degeneration and the formation of fibrous tissue.

Similar lesions are generally found in the spinal cord and its membranes. Cerebrospinal syndromes vary according to the character and location of the lesion, as well as the extent and duration, and express themselves in the form of convulsions, somnolence, hemiplegia, paraplegia, tetany, chorea, encephalitis, multiple sclerosis, aphasia, optic neuritis, and a variety of functional nervous and mental disorders.

In the heart, symptoms of coronary thrombosis, angina pectoris, heart block, and myocardial insufficiency may be produced; in the lungs, bronchitis, asthma and emphysema; in the gastro-intestinal tract, glossitis, gastritis, ulcer syndrome, constipation or diarrhea, cardiospasm, pylorospasm, enterospasm and anospasm; in the urogenital tract, occasional albuminuria, glycosuria, hematuria and vesicle tenesmus, as well as uterine bleeding, abortion and sterility in the female, and loss of libido and potentia in the male.

DISCUSSION OF CLINICAL SYNDROMES

As a result of careful clinical investigation in a series of 114 consecutive cases of chronic carbon monoxide anoxemia studied, I have found a classification into the following groups practical:

(1) Simple anoxemia
(2) Hematologic
(3) Cerebrospinal
(4) Cardiovascular
(5) Respiratory
(6) Gastro-intestinal

SIMPLE ANOXEMIC SYNDROME

This group is represented by the acute cases of asphyxiation and the milder chronic forms which terminate in recovery without any delayed or residual manifestations. The symptoms are limited to those resulting from oxygen deprivation, namely: headache, vertigo, paresthesia, stupor, dullness, yawning, dyspnea, palpitation, tremor, flushing, anorexia, nausea, vomiting, weakness, ataxia, myalgia, neuralgia, and so on. This anoxemic syndrome naturally forms an integral part of all the other syndromes to be described later.

The following family group is typical of the anoxemic syndrome:

Mr. and Mrs. P., of West Virginia, exhibited practically all of the symptoms enumerated: weakness, vertigo and a staggering gait being the most outspoken. Vertigo subsided and gait appeared normal on the third day of their stay at the clinic. Personal history revealed that they cooked and heated their home with natural gas, using open heaters not connected with flues. Their son, aged 3, slept in a room with gas burning all night. He was sickly, pale, lethargic, too weak to play, and vomited every second or third day. The baby, aged 1 year, who slept with the parents, suffered in a similar manner. Five canaries in one of the gas-heated rooms stopped singing when the cold weather set in, refused food, became shaggy and died. (Canaries were formerly used to detect dangerous amounts of the gas, as birds succumb more readily than human beings.) After the installation of proper heating appliances all symptoms subsided and since then all have enjoyed perfect health.

HEMATOLOGIC SYNDROMES

Two hematologic syndromes are clinically recognized: the polycythemic (which is quite common) and the anemic (which is rather rare). The following cases serve to illustrate both forms:

Case 1.—Mr. C., aged 29, West Virginia restaurateur, presented the foregoing anoxemic syndrome associated with a relative polycythemia. Chief complaints: weakness, headache, vertigo, nausea, dysphagia, unsteady gait, cough, dyspnea, neuromusclar pains and muscular fibrillation. Physical examination was negative except for obesity and absence of knee jerks. Laboratory studies were negative except for the blood examination. The hemoglobin was 110 per cent, red cells 7,830,000, leukocytes 10,200; polymorphonuclears, 68 per cent; small lymphocytes, 23 per cent; large lymphocytes, 8 per cent; eosinophils, 1 per cent. He was exposed day and night to escaping gas from stoves not connected with chimneys and frequently went out of doors for relief of his symptoms.

Cases 2 and *3.*—Mr. and Mrs. C., of West Virginia, both aged 62, were admitted to Mercy Hospital with symptoms characteristic of advanced Addisonian anemia. The history of onset, duration, clinical course, symptomatology and objective findings were identical in both

patients. Mr. C.'s blood showed 60 per cent hemoglobin, 1,766,000 red cells; Mrs. C.'s 64 per cent hemoglobin and 1,936,000 red cells. Both were achylic and complained of sore tongue in addition to profound weakness, paresthesia, headache, vertigo, stupor, palpitation and dyspnea. After two blood transfusions, Mrs. C.'s hemoglobin rose from 64 per cent to 80 per cent, and erythrocytes from 1,936,000 to 4,064,000. Her recovery was uneventful. Mr. C.'s response to the same treatment was not so satisfactory. He had a relapse after he returned home. A third transfusion was followed by a violent reaction from which he failed to rally.

The striking similarity suggested a common etiologic factor. After seemingly exhausting every effort to find a common cause, the following history was obtained: for eighteen years the family burned gas from their own well for cooking, and in open gas stoves for heating. During the winter months gas was burned continually day and night in unventilated rooms occupied by Mr. and Mrs. C. The children, who slept on the second floor without gas heat and with wide open windows, remained healthy. A dog allowed to occupy the first floor and to sleep in front of an open gas fire became ill and developed symptoms similar to those of Mr. and Mrs. C. Later he died from this illness.

DISCUSSION

By maintaining a relative carbon monoxide anoxemia in guinea-pigs, Nasmith[6] showed that a steady increase in erythrocytes occurred which reached a maximum in three or four weeks. Gerbis observed in dogs similarly treated that the hemoglobin and red cells, after reaching a maximum, began to decrease in two or three months, reaching normal at the latest in nine months. This phenomenon has been observed clinically in industrial workers daily exposed to gas. In forty-four patients of my series of ninety-seven analytically studied, the red cell count ranged from 5,000,000 to 7,800,000. In sixteen, the hemoglobin was 100 per cent or above. Comparatively few develop severe anemia, as it is apt to occur only after exposure for a number of years.

CEREBROSPINAL SYNDROMES

Carbon monoxide anoxemia preeminently affects the central nervous system. A few cases of the more pronounced forms of chronic cerebrospinal anoxemia are presented.

Case 1.—A married woman, aged 27, from West Virginia, suffered from typical attacks of tetany, chronic encephalitis and hypopituitarism. Cerebral tumor was suspected. History showed that the patient had been subject to attacks of transient numbness in the left forearm and hand for four years, and that these attacks became steadily worse during pregnancy. Two weeks before delivery she was profoundly stuporous and could not be easily aroused. When aroused she was confused and disoriented. She voided involuntarily with the attacks. The family noticed that during the period of stupor there was escaping gas in the room, which was heated by a gas heater without connecting flues. She was singularly free from attacks after the delivery of her child. Her nurse became ill with nausea, vomiting, headache, dizziness and drowsiness after the first night. Her mother, and later her father, who nursed her, were taken ill with headache, vertigo and nausea. At the end of the week the heating system was changed and the patient gradually emerged from the stupor with return of her tetany attacks. These subsided in July and she had no diurnal attacks until October. Examination showed a hypopituitary type with marked vasomotor disturbances. During spinal puncture a typical tetany attack developed. The impression was that the patient was suffering either from neoplasm in the right posterior central area, or encephalitis.

Case 2.—A farmer and stock dealer, aged 36, presented a history of coronary thrombosis, heart block and symptoms of chronic encephalitis with Parkinsonian syndrome. Personal history revealed that the family lived in the midst of a newly developed oil and gas field, and that at the time of his illness there were twenty-nine wells located within a radius of a mile, some within a quarter of a mile from his residence. These wells for a time were allowed to flow open, the gas escaping into the air. The gaseous vapor of some of these wells was piped up on a hill, away from the road to which it would drift and make traffic dangerous, and at times impassable. During the day the patient spent his time in watching the men drilling the wells. His house was heated by burning this unrefined wet gas day and night in open unventilated stoves. The heater in the bedroom in which he was confined during his illness was considered unsafe. His wife noticed the smell of gas and his doctors, also recognizing it, ordered the windows opened for protection. During this confinement he was stuporous, delirious, gasped for breath, broke out in cold perspiration, was nauseated and vomited.

The patient's wife became ill and suffered from nervousness, dizziness and headache. The sister who slept in an adjacent room was similarly affected.

A man engaged for two years in drilling wells was confined to bed, chronically ill with smothering, weakness, dizziness, hot flushes, and so on. Many neighbors were also affected.

Twelve sheep out of a flock of ninety-three died within two weeks after the patient came to the Clinic. They were grazing in a field adjoining a group of large wells. The sheep which survived were weak, trembled and staggered.

It is obvious that individuals residing in this environment suffered from the combined effects of benzene and its derivatives and carbon monoxide. The latter is not a constituent of natural gas, occurring only in consequence of incomplete combustion of the gas from the use of defective heaters. The former was no doubt responsible for the death of the sheep in pasture.

Case 3.—Symptoms of multiple sclerosis occurred in a woman 43 years of age with a relative polycythemia, vertigo, muscular weakness, ataxia and tendency to fall.

Upon inquiry as to the health of other members of her household, she replied: "We are all a bunch of inva-

lids, chronic complainers." This included father, mother, sister and aunt, all of whom were somewhat similarly affected, and about equally exposed in the home to gas from defective heaters and improper ventilation. The heater used in the patient's apartment was particularly defective and of a type condemned as unsafe seven years previously. An engineer stated that in the same county several deaths occurred from acute asphyxiation in homes where this type of heater was allowed to burn in the bedroom during the night.

After six weeks' treatment the following improvement was noted: a gain of twelve and one-half pounds, relief from headache, vertigo, paresthesia, muscle cramps and contractions of tendons of toes; diminution of palpitation, throbbing vessels and flushing, as also a gain in strength and steadier gait. The hemoglobin decreased from 103 to 86 per cent; the red cells from 5,600,000 to 4,300,000; blood cholesterol from 252 to 168 mg. per cent, while the basal metabolism increased from minus 13.2 per cent to plus 2 per cent.

DISCUSSION

The cases cited are only a few examples of the many forms of encephalopathies which may occur in chronic carbon monoxide anoxemia. Considering the nature and distribution of pathologic lesions in the central nervous system as demonstrated experimentally in animals by Yant[7] and his coworkers, and revealed at autopsy, it is easily comprehended why the symptoms are so numerous and protean in character. In this respect the condition closely resembles epidemic encephalitis, in which many bizarre and atypical forms have been described.

Carbon monoxide encephalitis of the Parkinsonian type frequently occurs owing to the fact that lenticular lesions are unusually common in these cases. Five well-defined cases of Parkinsonian encephalitis occurred in our series. Convulsive seizures of some sort, mostly epileptoid in character, were quite common.

Curious emotional episodes have been noted in some of the patients. They are usually accompanied with intense fear, during which the patient screams from fright.

Cases have been reported of temporary blindness, deafness, neuritis, gangrene, diabetes and psychosis, all of which can be regarded, in the light of our present knowledge, as purely symptomatic diagnoses representing a single expression of a more generalized disease. Disseminated sclerosis has been reported by McGurn[8] and others.[9] Putnam[10] and his associates produced patchy demyelinization by carbon monoxide. They attributed it to the formation of emboli or thrombi, causing a slight relative anemia.

In four cases brain tumor was either diagnosed or suspected. In none of them could the diagnosis be definitely established or sustained.

CARDIOVASCULAR SYNDROMES

The etiologic relation of anoxemia to cardiovascular diseases has not been sufficiently emphasized, and carbon monoxide as a factor in the production of anoxemia of the heart muscles may easily be overlooked. The following cases are cited as examples:

Case 1.—Mr. K., aged 59, of Oklahoma, was suffering from diabetes mellitus, oral sepsis, thyroid deficiency, and, for the past two years, from frequent attacks of precordial distress, clinically diagnosed angina pectoris. History revealed that the patient had no attacks of angina pectoris during the summer months. During November and December and until January 10, when he went to a Dallas clinic, attacks occurred daily. From Dallas he came to Baltimore for further study and observation, and it was observed that there were no attacks during his entire absence from home. Further inquiry disclosed that he had been exposed to carbon monoxide for more than twenty-five years; that natural gas was used in open heaters in his house, which was inadequately ventilated; that for the past five years he was exposed on the gas and oil fields; that he frequently detected escaping gas in his automobile while driving with the windows closed.

In addition to anginal attacks he suffered from soreness and weakness in the muscles of the legs, unsteady gait, cramps in the calf muscle, pressure type of headache, slight vertigo, dyspnea, cardiospasm and paresthesia. He used alcohol and tobacco moderately, drank coffee to excess, and overworked.

On the assumption that the anginal attacks were due to anoxemia of the heart muscle superinduced by carbon monoxide, and further influenced by a low basal metabolic rate, the patient was instructed to improve his personal hygiene by restricting alcohol, tobacco and coffee; to reduce his hours of work, to live in an atmosphere free from the possibility of escaping carbon monoxide gas, and at the same time to control his diabetes by diet, and his metabolism by the administration of thyroid. The last report (April, 1935) stated that he was free from angina pectoris.

Case 2.—A physician, aged 74, presented himself for examination in December, 1935, with the diagnosis of angina pectoris, asthma and bronchitis, and a history of coronary thrombosis fourteen years previously. He gave a history of having been exposed to carbon monoxide for thirty years, during which period he frequently experienced the symptoms characteristic of anoxemia. It is significant that during the summer months of 1935 he had no attacks of angina pectoris, while during the following winter months he had frequent attacks. The onset of attacks coincided with the seasonal use of gas in heating the office. Since last December, when he resorted to coal instead of gas for heating his office, he has been absolutely free from attacks.

DISCUSSION

These two cases (as well as *Case 2* reported as manifesting an encephalitic syndrome) are in-

structive in that the clinical observations confirm the view generally accepted that myocardial damage may result from anoxemia of the heart muscle and that it is one of the contributing factors in angina pectoris. The effect is the same whether the anoxemia is produced by carbon monoxide, severe anemia, polycythemia, or by high altitude. It has been observed that persons suffering with myocardial disease are very susceptible to carbon monoxide.

Herrick and Nuzum[18] first called attention to the relation of angina pectoris to anemia. Later Coombs,[20] Herrick[19] and Bullrich[21] observed that the severity of the angina was proportional to the degree of anemia (to the anoxemia). As the anemia improved, the angina tended to disappear. Angina pectoris occurs likewise occasionally in polycythemia vera and in myxedema, in which the association was first observed by Osler[23] and later confirmed by other investigators. In both conditions cellular metabolism is impaired owing to abnormal oxygen exchange.

RESPIRATORY SYNDROME

Carbon monoxide being a non-irritating gas, it does not produce severe inflammatory reactions in the lungs such as result from the irritating gases used in warfare. However, as hoarseness, cough and respiratory distress are frequent symptoms, tuberculosis and other chronic lesions may be suspected, as illustrated by the following case report:

A laborer, aged 51, suspected of having tuberculosis, complained of a chronic cough, and for ten days prior to examination of a constant uncontrollable yawning. The yawning developed while the patient was working in a gas-compressing plant in West Virginia and ceased before he had traveled half the distance from home to Baltimore, where he was being transported for examination. Prior to working in the gas-compressing plant he had been exposed for many years in the gas fields, although he never suffered from acute asphyxiation. He heated his house with natural gas by unvented heaters. He presented the symptoms characteristic of carbon monoxide anoxemia. Examination was negative for tuberculosis.

By improving heating conditions and avoiding undue exposure to gas, he was relieved of all his symptoms except some unsteadiness of gait.

Cough is a common symptom among those much exposed to gas. It is non-productive and usually relieved in a few days if the patient lives in an atmosphere free from carbon monoxide.

Hoarseness is often an annoying complaint. Frequent exposure as in the chronic form is said to predispose to asthma and bronchitis, while acute asphyxiation predisposes to pneumonia.

GASTRO-INTESTINAL SYNDROMES

Victims of carbon monoxide anoxemia frequently suffer from cardiospasm and pylorospasm combined with other gastric disturbances which lead one to suspect ulcer. Tendency to enterospasm associated with abdominal cramp and severe constipation suggests the possibility of perforation or intestinal obstruction, as indicated by the following case reports:

Case 1.—A man, aged 75, of West Virginia, had a history of gastro-enterostomy for perforated ulcer, which relieved symptoms only temporarily; continual periodic nausea and vomiting in addition to weakness, chilliness, cramps in the muscles of the thighs and feet, vertigo and staggering gait; and exposure to burning gas in a poorly ventilated office. From his symptoms and atmospheric environment, it was obvious that he was suffering from gas poisoning. Accordingly, he was advised to remain away from his office and to report at the clinic for gastro-intestinal studies. These studies proved to be negative. After three days in the clinic, his symptoms disappeared. He has been free of gastric symptoms since he has changed his office, which is now heated by a central heating plant.

Case 2.—A garage worker with symptoms of moderate gassing for several weeks was suddenly seized with an attack of dyspnea, abdominal pain and paroxysms of cramps, preceded by vomiting. Immediate operation was contemplated, as perforated ulcer was suspected, although the abdominal muscles, though tense, were not painful. After four hours the symptoms began to subside, the leukocytes dropped from 17,050 to 7,000, and the patient rapidly recovered without operation. Twenty-four hours after exposure the blood still showed 5 per cent saturation with carbon monoxide.

DISCUSSION

As was previously stated, anorexia, nausea and vomiting are common symptoms in anoxemia. In chronic anoxemic states, there is a tendency to subacidity and achylia. Cardiospasm and pylorospasm frequently occur.

Shumway[26] observed that foot traffic policemen complained more of gastric disorders than did those assigned to other duties. Their symptoms invariably subsided after a few days' rest. It would seem quite natural that these patients with a history of ulcer should have suspected a recurrence of the condition.

DIAGNOSIS

The diagnosis of chronic carbon monoxide anoxemia should not be difficult. The clinical syndrome of anoxemia is as characteristic of the condition as Korsakoff's is for a certain form of alcoholic neuritis, or Basedow's for exophthalmic goiter. The tendency has been to stress the secondary syndromes such as the Parkinsonian in encephalitis, Charcot's in multiple sclerosis,

the syndrome of angina pectoris, and many others, and regard them as primary factors when in reality they are often secondary manifestations of a chronic state of anoxemia.

Owing to the wide range of distribution of the lesions, in severe anoxemia several secondary syndromes may coexist in the same individual, as illustrated in some of the cases cited.

A relative polycythemia occurs in about 40 per cent of patients suffering with chronic carbon monoxide anoxemia. As the symptoms of the latter are essentially those of anoxemia accompanying states of anemia, the blood picture in these cases should make one suspect the disease, especially in the presence of a history of exposure and the effect of such exposure on the symptoms. Where natural gas is used for heating, the symptoms fluctuate with the seasons of the year.

Many of the milder cases are erroneously diagnosed neurasthenia or psychasthenia, owing to the absence of any very definite clinical findings. However, the blood morphology, bradycardia, hypotension, hypometabolism, gastric subacidity with tendency to glycosuria are contributory factors in making the diagnosis.

REFERENCES

1. Beck, H. G.: The Clinical Manifestations of Chronic Carbon Monoxide Poisoning. Ann. Clin. Med., **5**:1088-1096 (June) 1937.
2. Beck, H. G.: Slow Carbon Monoxide Asphyxiation: A Neglected Clinical Problem. J.A.M.A., **107**:1025-1029 (Sept.) 1936.
3. Sayers, R. R.; and Yant, W. R.: The Treatment of Carbon Monoxide Poisoning, Department of Interior, Bureau of Mines. Reports of Investigators, Serial No. 2476 (May) 1923.
4. Gerbis, Hermann: Zur Frage der chronischen Kohlenoxydvergiftung. Deutsche Med. Woch., 25:s. 991 (June 21) 1935.
5. Williams, Isabella R.; and Smith, Erna: Blood Picture, Reproduction and General Condition During Daily Exposure to Illuminating Gas. Amer. Jour. Physiol., **110**:611-615 (Jan.) 1935.
6. Nasmith, G. E.: The Hematology of Carbon Monoxide Poisoning. Jour. Physiol., London, **35**:32-52, 1906-7.
7. Yant, W. P.; Chornyak, J.; Schrenk, H. H.; Patty, F. A.; and Sayers, R. R.: Studies in Asphyxia. Public Health Bulletin No. 211.
8. McGurn, W. J.: Chronic Gas Poisoning. Interstate Med. Jour., **24**:479-487, 1917.
9. Mott, F. W.: Carbon Monoxide and Nickel Carbonyl Poisoning. Arch. Neurol., London County Asylums, **3**:262, London, 1907. (Cites Becker.)
10. Putnam, Tracy; Morrison, L.; and McKenna, John: Experimental Demyelinization. Trans. Amer. Neurol. Assn., **57**: 451, 1931.
11. Holm, K.: The Acute and Chronic Carbon Monoxide Poisoning. Med. Welt., **7**:191, 1933.
12. v. Bergmann, G.: Erstickung im Herzmuskel als Ursache der Angina Pectoris. Deutsche Med. Wchnschr., **60**:1378 (Sept.) 1934.
13. Keefer, C. S.; and Resnik, W. H.: Angina Pectoris: A Syndrome Caused by Anoxemia of the Myocardium. Arch. Int. Med., **41**:769 (June) 1928.
14. Allbutt, Sir Clifford: Diseases of the Arteries, Including Angina Pectoris. Ed. 1, vols. 1 and 2. New York: The Macmillan Company, 1915.
15. Williams, F. A.; and Giffin, H. Z.: The Anginal Syndrome in Pernicious Anemia. Amer. Jour. Med. Sci., **174**:30 (July) 1927.
16. Elliott, A. H.: Anemia as the Cause of Angina Pectoris in the Presence of Healthy Coronary Arteries and Aorta: Report of Case. Amer. Jour. Med. Sci., **187**:185-190 (Feb.) 1934.
17. Cabot, R. C.: Facts About the Heart, ed. 1. Philadelphia: W. B. Saunders Co., 1926.
18. Herrick, J. B.; and Nuzum, F. R.: Angina Pectoris. J.A.M.A., **70**:67 (Jan.) 1918.
19. Herrick, J. B.: On the Combination of Angina Pectoris and Severe Anemia. Trans. of the Assn. of American Physicians, **42**:23-28, 1927.
20. Coombs, C. A.: A Note on the Cardiac Symptoms of Pernicious Anemia. Brit. Med. Jour., **2**:185, 1926.
21. Bullrich, R. A.: Influencia pathogenica de los estados anemicos sobre la angina de pecho. Semana Medica (Buenos Aires), **2**:1137, 1925.
22. Kahn, M. H.: Etiologic Factors in Angina Pectoris. Amer. Jour. Med. Sci., **172**:195 (Aug.) 1926.
23. Osler, Sir William: Angina Pectoris as an Early Symptom in Aneurysm of the Aorta. M. Chron., **44**:11, 1906.
24. Means, J. C.; White, P. D.; and Krantz, I. C.: Observations on the Heart in Myxedema with Special Reference to Dilatation and Angina Pectoris. Boston Med. & Surg. Jour., **195**: 455, 1926.
25. Sturgis, C. C.: Angina Pectoris as a Complication in Myxedema and Exophthalmic Goiter. Boston Med. & Surg. Jour., **195**:351, 1925.
26. Shumway, E. A.: Chronic Carbon Monoxide Poisoning, an Increasing Danger to Municipalities from Automobile Traffic. Med. Jour. & Rec., **121**:657-659 (June) 1925.

ASTHMA IN HEART DISEASE*

A CLINICAL STUDY WITH ESPECIAL REFERENCE TO CARDIAC ASTHMA

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By far the majority of investigations into the subject of asthma in heart disease have been made by those whose primary interest was in heart disease. Pratt,[1] Palmer and White,[2] McGinn and White,[3] Weiss and Robb,[4] Harrison and his associates,[5] Longcope,[6] Kahn[7] and others have made the historical reviews, clinical descriptions, etiological, prognostic, therapeutic and pathologic-physiological observations upon which present concepts of cardiac asthma are based. McGinn and White[3] formulated the representative concept which follows:

"Cardiac asthma is a name applied to a kind of dyspnea peculiar to organic cardiac disease. For this particular condition it is distinctive and is preferable to such other terms as pulmonary edema or paroxysmal dyspnea, for it is truly asthmatic in nature and it is fundamentally of cardiac origin. Cardiac asthma is paroxysmal, coming on usually in sleep, but at times following exertion. An attack quickly rises to a peak, is accompanied by both inspiratory and expiratory difficulty, and frequently by a terrifying sense of suffocation which causes the patient to sit up or to stand erect or even to go to a window for air. The attacks last from a few minutes to a few hours, averaging about an hour,

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and leave the patient in an exhausted condition for hours or days. Adrenalin sometimes relieves the condition, but morphine and the upright position are nearly specific. Cardiac asthma is infrequently seen before middle life and is most commonly associated with cardiovascular conditions resulting in left ventricular enlargement and failure."

Weiss and Robb[4] and Harrison and his associates[5] have, on excellent evidence, described a mechanism which will explain paroxysmal nocturnal orthopnea in patients with heart disease. They have not explained, nor have they seriously attempted to explain, why some patients have the paroxysmal orthopnea with wheezing while others may have just as much orthopnea without wheezing. Harrison *et al* and several other authors do not consider wheezing a prerequisite to the diagnosis of cardiac asthma.

Contributions by those interested in allergy are few. Harkavy[8] reported four patients with "cardiac asthma" who were not relieved until allergenic influences had been removed. Walzer[9] has an unsatisfactory discussion of the subject. Rackemann[10], in an excellent discussion, predicates that:

"Even though the onset of asthma can be made to correspond with the onset of the heart disease, it can still be assumed that the particular patient has a predisposition to asthma." He observed also that, "if cardiac asthma were commonly dependent upon simple heart failure with pulmonary stasis, one would suppose that at least a large proportion of patients with heart disease would show asthma, but they do not."

The present study suggested itself when the routine allergic investigation of several patients, with what seemed to be cardiac asthma, disclosed that they had had mild wheezing with "colds" or from other known causes for years before the first conceivable evidence of impaired cardiac reserve. From this the tentative hypothesis was formulated that: wheezing in paroxysmal orthopnea is provoked non-specifically by the circulatory changes occurring in attacks of orthopnea, only in patients who are already asthmatic or in those already allergic in whom asthma is precipitated by the illness, heart disease. This will be discussed in some detail later.

This investigation was undertaken primarily to see what evidence could be obtained that allergy plays a role in the etiology of wheezing in paroxysmal orthopnea. A survey of the literature does not disclose a similar attempt, although White and McGinn[3] anticipated the need of it.

Selection of Cases.—The 47 cases of heart disease and wheezing, to be analyzed, were referred to us upon our request from the Medical Out-Patient Department and wards. Thirty of them were found among approximately 2,500 new patients of all kinds studied on the medical service in one year, an incidence of over 1 per cent. For comparison 14 patients with similar types and degrees of heart disease but without asthma were studied in the same way.

Method of Study.—The goal of each examination was to obtain all possible evidence of cardiovascular disease and of the various manifestations of allergy. Particular efforts were made to determine the date of onset of the first manifestation of cardiovascular disease and of asthma. This goal was not reached in all cases, but the data obtained were adequate for the purposes of this report.

The evidences of cardiovascular disease were obtained during the routine management of the patients by the various members of the medical staff. These details appear in the tables and on the whole are self-explanatory.

The evidences of the various manifestations of allergy were obtained in most instances by one of us whose chief interest is not in allergy, but who has worked in the Allergy Clinic long enough to conduct an adequate investigation. The allergy investigation consisted of a detailed history and skin tests. The skin tests were arbitrarily limited in most instances to intradermal testing with about twenty of the commonest allergenic foods and inhalants. The results are tabulated by the number of 1 plus, 2 plus and 3 plus reactions obtained. The history was much more detailed. First, was there a positive family history of allergy? Second, were there any other allergic manifestations than asthma? Third, what effects did the patient attribute to the common allergenic foods, inhalants and physical influences? The original records of the food histories contained many somewhat vague effects such as "indigestion," "disagrees with me," "causes gas." In the final tabulations of effects only the definite gastro-intestinal symptoms listed in the footnotes of the tables and distant allergic manifestations were included. The effects attributed to contacts with allergenic inhalants such as dusts, smoke, epidermals, fumes, powders and pollens do not require comment. The effects of those physical influences such as exercise, fatigue and excitement which are also provocative of cardiac symptoms were not included in the tables. In most cases the effects attributed to physical influences were provoked by damp weather, sudden temperature changes,

excessive heat or cold, draughts, or getting wet by rain.

The question of the duration of the manifestations of cardiovascular disease and of asthma is most important. Accordingly every effort was made to date the first knowledge of hypertension or of dyspnea or palpitation, or of anginal pain. It was relatively easy to determine the time of onset of the symptoms referable to congestive failure or coronary occlusion. Leading questions were used freely in the effort to date the onset of asthma. The same approach that is used in the Allergy Clinic was used in these cases, namely: "When was the first time that you noticed wheezing?" "Do you wheeze with chest colds?" "How long have you been doing so?" "When you had 'flu' in 1918 did you wheeze and have to sit up to get your breath?" and so on.

Analysis of Data.—Preliminary analyses of the information gathered as outlined above suggested certain criteria by which the cases could be subdivided for more detailed study. The chief criterion selected was the severity of the asthma. All cases with asthma of sufficient severity to cause them to seek relief were grouped (*Table 1*) under the heading of troublesome asthma. Those cases with asthma as an incidental finding and accepted by the patients as of little importance were put in the group (*Table 2*) having slight, not troublesome, asthma. A third group (*Table 3*) was made up of those cases of congestive and/or anginal failure in whom no history or physical signs of asthma could be elicited. These served as controls.

More detailed study of the cases in Table 1 showed that there were some (1 to 19) in which asthma had begun many years before the first symptom referable to heart disease. There were others (20-25) in which there was reasonable doubt as to the priority of the asthma or of the heart disease. And in a third group (26-28) there were no physical signs of asthma though there was the history of wheezing.

The first group of cases in this table was further subdivided into those (1-10) which had congestive failure, those (11-14) which did not have congestive failure and those (15-19) with several manifestations of cardiovascular syphilis with or without congestive failure. Additional details of the varied evidences of cardiovascular disease are pointed out under remarks in the table. There was a striking uniformity in the clinical courses of these cases. In each there was the history of mild wheezing with colds or from other known causes for many years. With the advent of symptoms of significant lowering of the cardiac reserve they began to have typical severe attacks of nocturnal paroxysmal dyspnea with wheezing, in other words, cardiac asthma. The known causes of asthma in each of the tables are classified, but not detailed, under the heading "history of effects of common excitants." It will be noted that with one exception (Case 9) there was at least one other allergic manifestation and with three exceptions there were at least two recognized causes of asthma. The allergic background and the long priority of the asthma do not seem open to question. It is interesting that there was a long period of freedom from asthma (Cases 3, 10 and 14) which lasted from young manhood until the onset of heart failure, when it became severe. Coronary occlusion initiated the severe asthma in Cases 9 and 11. Symptomless hypertension preceded cardiac symptoms and severe paroxysmal dyspnea by over ten years twice (Cases 9 and 13).

The group (20-25) in which there was reasonable doubt as to the priority of asthma needs some elaboration.

Case 20 had mild asthma with attacks of bronchitis two or three times a year for over ten years. He had been somewhat dyspneic on exertion for about six years. The asthma had been getting slightly worse for two or three years, but did not assume the proportions of cardiac asthma until he had mild congestive failure six months before he was studied. In addition to the asthma which, from the history, could be induced from certain foods and physical influences, he had a positive family history, a number of positive skin reactions, and some food idiosyncrasies. He died shortly after he left the hospital in an acute attack of congestive failure. His physician reported that there was no asthma during the final illness.

Case 21 had hay fever from known causes for about two years before the first sign of wheezing, which was followed in six months by dyspnea on exertion. The first observation was during a series of attacks of severe paroxysmal dyspnea, which eventuated in pulmonary edema. During these attacks there was little subjective wheezing. Digitalis relieved all of her cardiac symptoms. Before and after digitalization wheezing was noted only with colds and was unaffected by the state of her cardiac reserve. The family history was negative, as were the skin tests, and she had no other allergic manifestation but hay

Table 1

HEART DISEASE AND TROUBLESOME ASTHMA

Miscellaneous Data				Evidences of Heart Disease													Evidences of Allergy													Duration in Years	
				Failure		Rhythm		Electrocardiogram			Blood Vessel		Other Evidences					Other Allergic Manifestations						History of Effects of Com. Excit.			No. Pos. Skin Tests				
Case Number	Age	Race	Sex	Congestive	Anginal	Regular	Type of Arrhythmia	Myocardial Damage	Heart Block	Axis Deviation	Arteriosclerosis	Aortitis	Hypertrophy	Blood Pressure	Diastolic Murmur	Family History	Hay Fever	Food Idiosyncrasy	Migraine	Urticaria	Angioneurotic Edema	Eczema	Foods	Inhalants	Physical Influences	1+	2+	3+	Wheezing	Evidences of Heart Disease
Group I—See remarks below																														
1	66	W	F	2+	0	0	Fib	+	0	0	+	0	2+	250:125	0	+	+	+	+	0	0	0	0	HF Ast	HF	3	0	0	18	2
2	75	W	M	1+	+	+	0	+	0	0	+	0	0	210/110	0	+	+	0	0	0	0	0	0	Ast	0	2	0	0	30	6
3	41	C	M	3+	0	0	0	+	0	+	0	0	3+	150/114	0	+	+	0	0	0	0	0	0	Ast	Ast				25	1
4	37	W	M	3+	0	0	Fib.	+	0	0	0	0	2+	138/96	+	0	0	+	+	0	0	0	GI	Ast	Ast	3	1	1	30	6
5	65	W	M	2+	+	0	Gal	0	+	0	+	0	0	135/95	0	+	0	+	0	+	0	0	0	Ast	0	2	1	2	15	3
6	26	W	F	2+	0	0	Gal	0	0	0	0	0	2+	170/114	0	+	+	+	0	+	0	0	GI	HF Ast	0	9	2	0	20	1
7	59	W	M	4+	0	0	Fib	0	0	0	+	0	4+	220/80	0	+	0	0	+	+	0	0	Urt	Ast	Ast	0	1	0	25	8
8	52	W	F	1+	0	+	0	+	0	+	0	0	2+	235/130	0	+	+	+	0	+	0	+	Urt GI	HF Ast	HF Ast	0	0	0	10	6
9	56	W	M	2+	+	+	0	+	0	+	+	0	2+	205/135	0	0	0	0	0	0	0	0	0	Ast	Ast	2	0	0	18	8
10	66	W	M	1+	+	+	0				+	0	1+	220/110	0														38	7
Group II—See remarks below																														
11	59	W	M	0	+	+	0	0	0	+	+	0	1+	170/112	0	+	0	+	+	0	0	0	Ast GI	Ast	Ast				18	1
12	57	W	M	0	0	0	Gal Ex. Sys				+	0	1+	190/120	0	0	+	+	+	0	0	0	GI	HF	Ver Hea	3	2	0	21	1½
13	50	C	M	0	0	+	0				+	0	4+	210/115	0	+	0	0	0	+	0	0	Ast	Ast	0	4	0	0	20	15
14	65	W	M	0	0	0	Ex. Sys	+	0	0	+	0	1+	190/120	0	0	+	0	0	0	0	0	0	Ast	Ast	4	1	2	55	2
Group III—See remarks below																														
15	62	C	M	2+	+	+	0				+	+	2+	180/70	+	0	0	+	0	0	0	0	GI	Ast	Ast	5	0	1	18	6
16	57	W	M	1+	+	0	Flu Fib	0	0	0	+	+	2+	120/100	0	+	+	0	0	0	0	0	0	HF Ast	HF Ast	6	3	1	27	3
17	63	C	M	4+	0	+	0	+	0	0	+	0	1+	170/88	0	+	+	0	0	0	0	0	0	Ast	HF	0	0	0	35	9
18	56	W	M	0	0	+	0				+	+	0	140/95	0	+	+	0	+	0	0	+	0	HF	Ast	0	4	0	35	3
19	56	C	M	4+	0	+	0	0	0	+	+	+	3+	185/135	+	+	0	+	+	0	0	0	Hea GI	Ast	Ast	3	2	0	18	4

Table 1 (*Concluded*)

HEART DISEASE AND TROUBLESOME ASTHMA

Miscellaneous Data				Evidences of Heart Disease												Evidences of Allergy													Duration in Years	
				Failure		Rhythm		Electrocardiogram			Blood Vessel		Other Evidences				Other Allergic Manifestations							History of Effects of Com. Excit.			No. Pos. Skin Tests				
Case Number	Age	Race	Sex	Congestive	Anginal	Regular	Type of Arrhythmia	Myocardial Damage	Heart Block	Axis Deviation	Arteriosclerosis	Aortitis	Hypertrophy	Blood Pressure	Diastolic Murmur	Family History	Hay Fever	Food Idiosyncrasy	Migraine	Urticaria	Angioneurotic Edema	Eczema	Foods	Inhalants	Physical Influences	1+	2+	3+	Wheezing	Evidences of Heart Disease
Group IV—See text and remarks below																														
20	55	W	M	1+	0	0	Gal	+	0	+	+	0	1+	225/120	0	+	0		0	0	0	0	Ast GI	0	Ast	4	1	1	10	6
21	61	C	F	4+		0	Ex. Sys	0	0	+	+	0	4+	220/126	0	0	+	0	0	0	0	0	0	HF	0	0	0	0	2¼	2¼
22	57	W	F	1+	+	+	0	0	0	+	0	0	2+	212/132	0	+	+	+	+	0	0	0	Ast GI	HF Ast	0	1	0	1	3	½
23	50	W	F	1+	+	0	Gal	+	0	0	+	0	2+	200/138	0	+	0	+	0	0	0	0	0	Ast	Ast	3	1	1	7	6
24	52	W	M	4+	0	+	0	+	+	0	+	0	2+	160/70	0	+	+	0	0	0	0	0	0	HF Ast	Ast				1	1½
25	41	C	F	0	0	0	0	+	+	0	0	0		180/110	0	0	+	+	0	0	0	+	GI	0	Ast	3	0	1	1½	1½
Group V—See text and remarks below																														
26	65	W	M	1+	+	+	0	+	0	+	+	0	2+	180/90	0	0	+	0	0	0	0	0	0	HF	HF Ast	9	0	1	18	4
27	61	W	M	1+	+	0	Gal	+	0	0	+	0	2+	154/90	0	+	+	0	0	0	0	0	0	Ast	Ast	0	5	2	3	¼
28	65	W	M	2+	+	0	Alt Gal	+	0	+	+	0	1+	230/140	0	0	0	0	0	0	0	0	0	0	0				2	2

Table 1

Each case in the first three groups (1 to 19) had mild wheezing with colds and/or from other known causes for years. Wheezing was markedly aggravated by and was accompanied by attacks of paroxysmal orthopnea since the advent of symptoms of heart failure. See text for discussion.

Group I is made up of cases which showed various degrees of congestive failure in hypertensive and/or arteriosclerotic heart disease with coronary occlusion (No. 9), chronic nephritis with uremia (No. 6), arrhythmias, EKG changes, anginal symptoms and diastolic murmurs, as indicated.

Group II consists of hypertensive and arteriosclerotic heart disease without congestive failure. Coronary occlusion (No. 11) and other evidences of heart disease are as indicated.

Group III is made up of cases of cardiovascular syphilis. Cases 15 and 16 had aneurysms. Case 18 had markedly lowered cardiac reserve without congestive failure. Other evidences of heart disease are as indicated.

Group IV is made up of cases of hypertensive heart disease and asthma. The priority of asthma here is questionable. Note the incidence of other allergic phenomena, and of hay fever and asthma from common allergic excitants. See text for discussion.

Group V contains cases of typical paroxysmal orthopnea with the history of wheezing. No auscultatory wheezing was observed in Cases 26 and 27 during mild attacks. No auscultatory wheezing was heard between attacks in Case 28. Note evidences of allergic background in Cases 26 and 27 and their absence in Case 28.

In this and in subsequent tables blank spaces indicate that those observations were not made.

Abbreviations:

Fib. = Auricular fibrillation.
Gal. = Gallop rhythm.
Ex. Sys. = Extra systoles.
Flu. = Auricular flutter.
Alt. = Pulsus alternans.
H. F. = Hay fever.
Ast. = Asthma.
G. I. = Abdominal pain, nausea, vomiting, heart burn, diarrhea, or constipation.
Ver. = Vertigo.
Hea. = Headache.

Table 2

CONGESTIVE AND ANGINAL FAILURE WITH SLIGHT (NOT TROUBLESOME) ASTHMA

Miscellaneous Data				Evidences of Heart Disease												Evidences of Allergy													Duration in Years	
				Failure		Rhythm		Electrocardiogram			Blood Vessel		Other Evidences			Family History	Other Allergic Manifestations						History of Effects of Com. Excit.			No. Pos. Skin Tests				
Case No.	Age	Race	Sex	Congestive	Anginal	Regular	Type of Arrhythmia	Myocardial Damage	Heart Block	Axis Deviation	Arteriosclerosis	Aortitis	Hypertrophy	Blood Pressure	Diastolic Murmur		Hay Fever	Food Idiosyncrasy	Migraine	Urticaria	Angioneurotic Edema	Eczema	Foods	Inhalants	Physical Influences	1+	2+	3+	Wheezing	Evidences of Heart Disease
Group I—See remarks below																														
1	55	C	M	1+	0	+	0				+	0	1+	175/95	0	+	+	0	0	0	0	0	0	HF Ast	Col	0	0	0	18	9
2	53	W	M	1+	+	+	0		0	0	+	0	1+	198/128	0	+	+	0		0	0	0	GI	HF	0	6	4	0	18	1
3	58	W	M	1+	+	+	0	+	0	0	+	0	1+	155/110	0	+	+	0	0	0	0	0	0	HF	Col	5	2	1	15	1½
4	15	W	F	4+	0	0	Gal	+	0	0	0	0	4+	170/110	0	+	0	0	0	0	0	0	0	Ast HF	0	0	1	1	20	3
5	42	W	F	4+	0	0	Fib	0	0	0	0	0	2+	170/114	+	+	+	+	0	0	0	0	GI	Ast	Ast	3	0	0	17	3
6	61	C	M	2+	0	0	Gal	0	0	0	+	0	2+	230/140	0	+	+	0	+	0	0	0	Ast	HF Ast	0	0	0	0	40	6
7	50	W	F	1+	0	0	Ex. Sys.	0	+	0	0	0	2+	204/106	0	0	+	+	+	0	0	0	Urt Ast	Ast HF	0	0	0	0	18	6
8	69	W	M	0	+	+	0	+	0	0	+	0	1+	170/130	0	+	+	+	0	0	0	0	JT Pal	Ast	0				50	2
9	69	W	M	0	+	+	0				+	0	1+	220/140	0	+	0	0	+	+	0	0	0	0	Ast				45	½
Group II—See remarks below																														
10	73	W	F	1+	+	0	Ex. Sys.	0	0	+	+	0	2+	220/130	0	+	0	0	+	0	0	+	0	Ast	Hoa Hea					18	18
11	62	C	M	4+	0	+	0	+	0	0	+	0	1+	155/110	0	0	+	+	0	0	0	0	Ast	HF Ast	Ast				1	1
12	65	C	M	1+	0	0	Ex. Sys.				+	0	2+	200/118	0	+	0	+	+	0	0	0	GI	0	Col	1	0	0	2½	2/3
13	44	C	F	0	0	+	0				0	0	1+	188/142	0	+	+	0	0	0	0	0	0	HF	Ast	1	0	0	2	0
14	75	C	F	4+	0		0				+	0	2+	160/80	0	+	+	+	+	0	0	0	GI	HF	Col Ast				2/3	1
15	63	W	F	2+	0	0	Fib	0	0	0	+	0	3+	230/150	0	+	0	0	+	0	0	0	0	0	0	0	0	0	3	3½
16	76	C	F	4+	0	+	0	0	0	+	+	0	4+	200/80	+	0	+	0	+	0	0	0	0	Ast	HF	0	0	0	1/12	2/3
17	57	W	M	4+	0	+	0	+	0	0	+	0	1+	200/114	0	+	0	0	+	+	0	0	0	Hea Ast	Col	7	2	3	2	2
18	55	C	M	1+	0	+	0	0	0	0	+	0	1+	260/145	0	0	0	+	+	0	0	0	GI	0	0				¼	¼
19	50	C	M	4+	0	0	Fib	0	0	0	+	0	2+	210/110	0	+	0	0	0	0	0	0	0	GI	Col	0	1	0	1	1

Table 2 (*Concluded*)

CONGESTIVE AND ANGINAL FAILURE WITH SLIGHT (NOT TROUBLESOME) ASTHMA

	REMARKS
Group I	
Long-standing mild asthma occurring rarely except with colds or from known causes, not aggravated by the advent of anginal symptoms, coronary occlusion (Nos. 3 and 8), congestive failure, arrhythmias, chronic nephritis (Nos. 4 and 9), thyrotoxicosis (No. 7), lowered cardiac reserve (No. 9), lues (positive Wassermann Nos. 1 and 2) in hypertensive heart disease.	
Group II	
Questionable priority of wheezing and symptoms of heart disease. In Cases 10 to 15 wheezing is brought on by allergenic, non-cardiac agencies. Each has hay fever or allergic family history, or other allergic manifestations. In Cases 16 to 19 wheezing occurs only with "colds," independently of symptoms of congestive failure. The background of allergy is not as impressive in these. Active tuberculosis was present in Case 19.	

Abbreviations same as Table 1, plus:

Col = Colds. Hoa = Hoarseness. JT Pai = Joint pain

fever. This, however, is sufficient to classify her as an allergic individual.

Case 22 had asthma and hay fever from known foods and inhalants for two years before the onset of cardiac symptoms. The asthma is unaffected by the cardiac symptoms and rarely occurs except from known causes. There are multiple additional manifestations of allergy and two positive skin tests. This seems to be a clear-cut case of allergic asthma with hypertensive heart disease and congestive failure.

Case 23 had hay fever for many years. Moderately troublesome wheezing which began about seven years ago was greatly intensified by mild congestive failure and diagnosed as cardiac asthma a year later. Since then multiple physical and inhalant causes have been demonstrated in the Allergy Clinic. She has had no more congestive failure and very few troublesome attacks of asthma. She has in addition a positive family history and multiple food idiosyncrasies and several positive skin tests, obviously an allergic individual.

Case 24 had bronchiectasis for seven years and hay fever for four years. Mild asthma appeared after an attack of influenza and was markedly intensified after he developed congestive failure. He had a positive family history and known food and inhalant causes for his hay fever and asthma. Skin tests were not done. This, too, seems to be a case of asthma from non-cardiac causes, markedly accentuated by congestive failure.

Case 25 had asthma, hay fever, food idiosyncrasies, and eczema; hyptertensive heart disease with gallop rhythm, but no physical signs of failure. She also had a positive Wassermann, but no x-ray evidence of aortitis and no physical evidence of valvular disease. There were known physical causes of asthma. This patient with multiple allergic manifestations did not develop asthma until symptoms of lowered cardiac reserve appeared. We were unable to establish or disprove a definite relationship between the heart disease and the asthma.

In this group of cases there is abundant evidence of the allergic basis for the asthma in all but Case 21, in whom the asthma occurred independently of cardiac symptoms and only with colds. In Cases 20, 23 and 24 the asthma was markedly affected by the cardiac failure. In Cases 21 and 22 there was little or no aggravation of the asthma by the heart disease. In Case 25 the relationship was not established.

The last group in *Table 1* is included to show: first, that paroxysmal dyspnea which fulfills the

present requirements of some authors[5] for the diagnosis of cardiac asthma can occur without wheezing (Cases 26 and 27) even in patients who have the history of mild hay fever and wheezing from known causes for many years. Second, that paroxysmal dyspnea and the history of wheezing can occur in a patient (Case 28) with a completely negative family and personal history of any manifestation of hypersensitiveness. This patient was not examined during any of the typical attacks of cardiac asthma, but repeated examinations over a period of three years failed to elicit a single asthmatic sign. It is impossible, therefore, to say whether this patient had simply marked paroxysmal dyspnea and interpreted it as wheezing or asthma, or, whether he was the only non-allergic patient in the entire series who had true cardiac asthma with wheezing.

Little need be added to the remarks in *Table 2*, which contains the cases of mild asthma with various types and degrees of heart disease. The first group contains those with long priority of asthma which, in contrast to the comparable group in *Table 1*, was unaffected by the various degrees of congestive and anginal failure. The second group, comparable to those in *Table 1*, consists of those with questionable priority of asthma and symptoms of heart disease. Inasmuch as none of these patients had wheezing of sufficient severity to suggest cardiac asthma, more detailed discussions are not necessary. Attention should be called, however, to the high incidence of positive family histories, other allergic manifestations, positive skin tests, and known causes of hay fever, asthma, and other symptoms. *Table 3* is included simply to show, as is well known, that a variety of types and degrees of heart disease can occur without wheezing. The low incidence of the various evidences of allergy is striking.

DISCUSSION

Analysis of the data in the tables suggests a number of observations which seem highly pertinent to the subject of asthma in heart disease. The detection of 28 cases fulfilling the existing criteria for the diagnosis of cardiac asthma, and the concurrence of asthma and severe heart disease in nineteen additional cases, would seem to indicate that so-called cardiac asthma and the incidence of asthma and heart disease are not so rare as available statistics indicate.[2 11]

This relatively high incidence of asthma and heart disease creates the need of criteria for the classification of such cases. The criteria used in grouping the data in the tables seem adequate and practical. The chief point of difference was whether the asthma was troublesome or not troublesome. In each group there were some in which asthma had preceded the first symptom of heart disease by many years and others in which the onset of asthma and cardiac symptoms almost coincided. Further classification in the troublesome group was based upon the presence or absence of congestive failure or of cardiovascular syphilis. An additional potential criterion for further subdivision is whether or not the asthma is aggravated by the advent of cardiac symptoms.

The high incidence of hypertension, lues, gallop rhythm, auricular fibrillation, myocardial damage, anginal symptoms and cardiovascular syphilis, and the low incidence of mitral stenosis conforms to the observations of others already referred to. It seems appropriate to observe that the prognosis of cardiac asthma, as stated by White and McGinn[3] and others, probably does not differ materially from the prognosis of patients who have comparable degrees of heart disease, but who do not have cardiac asthma.

On the basis of our observations we feel justified in formulating the following concept of cardiac asthma. Wheezing is the fundamental and invariable sign of asthma. Without it asthma is not present. With it asthma is present. With well known exceptions, wheezing is usually attributed to an allergic reaction and is mediated by smooth muscle spasm plus exudation. The concept of Vaughan[11] that allergic manifestations can be provoked by non-specific causes such as fatigue, excitement, acute infections, emotional strain or temperature changes in a person who is in so-called "allergic equilibrium" has been widely accepted by allergists. It would seem logical, then, to assume that a person with serious heart disease who had paroxysmal nocturnal dyspnea would wheeze only if the mechanism for wheezing were previously established. The mechanism for wheezing is obviously established in patients who have had asthma. It is well known that patients with the capacity for sensitization tend to exhibit their first allergic manifestations after some trying emotional episode or illness. It is quite conceivable that lowering of the cardiac reserve is the determining factor in the appearance of asthma or wheezing in allergic individuals. These thoughts plus the marked exacerbation of previously mild wheezing in the first nineteen cases in *Table 1*, the high incidence

Table 3

CONGESTIVE AND ANGINAL FAILURE—NO ASTHMA—CONTROL CASES

Miscellaneous Data				Evidences of Heart Disease													Evidences of Allergy													Duration in Years	
				Failure		Rhythm		Electrocardiogram			Blood Vessel		Other Evidences					Other Allergic Manifestations						History of Effects of Com. Excit.			No. Pos. Skin Tests				
Case No.	Age	Race	Sex	Congestive	Anginal	Regular	Type of Arrhythmia	Myocardial Damage	Heart Block	Axis Deviation	Arteriosclerosis	Aortitis	Hypertrophy	Blood Pressure	Diastolic Murmur	Family History	Hay Fever	Food Idiosyncrasy	Migraine	Urticaria	Angioneurotic Edema	Eczema	Foods	Inhalants	Physical Influences	1+	2+	3+	Wheezing	Evidences of Heart Disease
1	21	W	M	1+	+		Fib	0	0	0	0	0	0	124/72	0	+	+	0	+	+	0	0	Urt	HF	Col	4	0	0	0	2
2	49	W	F	1+	+		Par Tac				0	0	2+	260/160	0	0	0	+	+	0	0	0	Mig Pai	Cou	0	4	0	0	0	1
3	58	C	M	1+	0		Ex. Sys	0	0	+	0	0	4+	200/130	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	1/12
4	56	W	M	0	+	+					0	0	2+	220/120	0	0	0	0	0	0	0	0	0	0	0	1			0	2
5	65	W	F	4+	0	+		+	0	0	+	0	4+	250/130	0	0	0	0	0	0	0	0	0	0	0				0	2
6	50	C	F	4+	0		Ex. Sys	+	0	0	0	0	4+	230/130	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	2
7	57	W.	M	4+	+	+		+	0	0	0	0	1+	150/110	0	0	0	0	0	+	0	0	0	0	0	1	1	1	0	1½
8	46	C	F	4+	0		Fib	+	0	0	+	0	4+	190/150	0	0	0	0	0	0	0	0	0	Cou	0	0	0	0	0	½
9	63	W	M	4+	+		Gal				+	0	3+	140/112	0	0	0	0	0	0	0	0	0	0	Col				0	1
10	53	W	M	4+	+		Fib	0	0	0	0	0	4+	130/94	0	+	0	0	+	0	0	0	0	0	0				0	21
11	28	W	M	4+	0		Gal	+	0	0	0	0	3+	154/65	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	9
12	58	W	M	4+	+	+		+	0	0	+	0	0	130/88	0	0	0	0	0	+	0	0	0	Cou	Cou	1	0	0	0	¼
13	73	C	M	4+	+	+		+	0	0	+	+	3+	160/65	+	0	0	0	0	+	0	0	Urt	0	0				0	2/3
14	42	C	M	2+	0		Gal				0	+	2+	160/45	+	0	0	0	+	0	0	0	0	0	0				0	1/12

REMARKS

No history or physical signs of asthma in these cases. Other allergy as indicated.

Cases 1, 2 and 3 had slight congestive failure. Case 1 was postoperative thyrotoxicosis with anginal failure. Case 2 was hypertensive heart disease with anginal failure and paroxysmal tachycardia. Case 3 was hypertensive heart disease.

Cases 4 through 13 had severe congestive failure. Among these were hypertensive heart disease with coronary occlusion, anginal symptoms, arteriosclerosis, various arrhythmias, and chronic nephritis. There were also in this group one case of luetic aortitis, one of adhesive pericarditis and another of postoperative thyrotoxicosis.

Case 14 was luetic aortitis and valvulitis with moderate congestive failure.

Abbreviations same as Tables 1 and 2, plus:

Par Tac = Paroxysmal tachycardia.

Pal = Palpitation.

Mig = Migraine.

Cou = Cough.

of known non-cardiac causes of asthma in these and in the next six cases, the high incidence of family and personal histories of other allergic manifestations in the *Tables 1* and *2* as contrasted with the non-asthmatic cases in *Table 3* certainly support the theory that: wheezing with paroxysmal nocturnal dyspnea is in the vast majority of cases an indication that the patient has previously had asthma of some degree, or that he is an allergic individual in whom the onset of asthma is provoked by the illness represented by loss of cardiac reserve, and that the changes[5] incident to the attack of dyspnea are the so-called "non-specific exciting factors" which initiate the wheezing.

This theory does not in any way discredit, but should be considered supplementary to, the current ones,[4 5] which are deficient only in that they do not explain why some patients with paroxysmal orthopnea wheeze while others do not.

This theory also emphasizes the importance of the sign wheezing. It is impossible to tell, in our experience, how much of the wheezing and dyspnea, in an attack or a series of attacks, is the non-specific cardiac effect or how much is allergic. This leads to the obvious and somewhat platitudinous advice that: inasmuch as paroxysmal dyspnea and asthma may each intensify the other, the proper approach to the problem of the patient who does not get complete (auscultatory) relief from current methods of cardiac therapy,[3 4] is to subject him to a detailed allergic investigation, not just a few routine skin tests. It should be mentioned here that proper cardiac management will control, very satisfactorily, a majority of cases of cardiac asthma.

The allergist hardly needs his attention called to the fact that the "non-specific exciting factor," the paroxsymal dyspnea syndrome, need not intensify the pre-existing or concurrent asthma. He is conversant with the wide variation in individual susceptibility of the allergic patient to these influences. This is illustrated extensively in *Table 2* and in Cases 26 and 27 of *Table 1*. Case 28 in *Table 1* indicates that it is not always possible to substantiate the opinions expressed above. This does not detract, we think, from the applicability of the theory to the large majority of cases of so-called cardiac asthma.

Finally, we wish to urge that the term cardiac asthma be limited only to those patients in whom asthmatic wheezing can be heard and in whom the "non-specific exciting factor," the paroxysmal dyspnea syndrome, can be shown to be more important, after adequate study, than the specific allergenic factors.

SUMMARY

(1) Twenty-eight cases of troublesome asthma, nineteen cases of mild asthma, and fourteen cases with no asthma, all with varying degrees and types of cardiovascular disease, have been studied.

(2) Evidence is offered that in 21 of 28 cases (*Table 1*) fulfilling the existing requirements for the diagnosis of cardiac asthma, the asthma antedated, in mild form, the nocturnal paroxysmal orthopnea by many years.

(3) Evidence is offered to support the theory that: the addition of wheezing to paroxysmal nocturnal dyspnea is in the large majority of cases an indication that the patient has previously had asthma of some degree, or that he is an allergic individual in whom the onset of asthma is provoked by the illness represented by loss of cardiac reserve, and that the changes[5] incident to the attack of dyspnea are the so-called "non-specific exciting factors" which initiate the wheezing.

(4) Criteria for the classification of cases which have asthma and heart disease concurrently are stated.

(5) Reasons are given for the necessity of a detailed allergic investigation in those cases which are not entirely relieved by current methods of treating so-called cardiac asthma.

(6) It is urged that the term cardiac asthma be limited only to those in whom asthmatic wheezing can be heard and in whom the "non-specific exciting factor," paroxysmal dyspnea, can be shown to be more important than the specific allergenic factors.

BIBLIOGRAPHY

1. Pratt, J. H.: Cardiac Asthma. J.A.M.A., **87**:809, 1926.
2. Palmer, R. S.; and White, P. D.: Clinical Significance of Cardiac Asthma. J.A.M.A., **92**:431, 1929.
3. McGinn, S.; and White, P. D.: Follow-Up Report; 250 Cases of Cardiac Asthma. New Eng. Jour. Med., **207**:1069, 1932.
4. Weiss, S.; and Robb, G. P.: Cardiac Asthma. J.A.M.A., **100**:1841, 1933.
   Treatment of Cardiac Asthma. Med. Clin. N. Amer., **16**: 961, 1933.
5. Harrison, T. R., *et al:*
   Congestive Heart Failure: XVIII. Clinical Types of Nocturnal Dyspnea. Arch. Int. Med., **53**:561, 1934.
   Congestive Heart Failure: XIX. Reflex Stimulation of Respiration. Arch. Int. Med., **53**:724, 1934.
   Congestive Heart Failure: XX. Cheyne-Stokes Respiration as the Cause of Paroxysmal Dyspnea at the Onset of Sleep. Arch. Int. Med., **53**:891, 1934.
   Congestive Heart Failure: XXI. Observations Concerning the Mechanism of Cardiac Asthma. Arch. Int. Med., **53**:911, 1934.
   Failure of the Circulation, Chapter 18. Baltimore: Williams & Wilkins, 1935.
6. Longcope, W. T.: Syphilitic Aortitis: Its Diagnosis and Treatment. Arch. Int. Med., **11**:14, 1913.

7. Kahn, M. H.: Cardiac Asthma. Amer. Ht. Jour., **2**:424, 1927.
8. Harkavy, J.: Bronchial Asthma Complicating Cardiovascular Disease. J.A.M.A., **82**:100, 1924.
9. Coca, A. F.; Walzer, M.; and Thommen, A. A.: Asthma and Hay Fever, p. 244. Springfield, Ill.: Chas. C. Thomas, 1931.
10. Rackemann, F. M.: Clinical Allergy, pp. 415-20. New York: Macmillan Company.
11. Vaughan, W. T.: Role of Specific and Non-Specific Factors in Allergy and Allergic Equilibrium. Jour. Lab. & Clin. Med., **13**:633, 1928.

DISCUSSION (*Abstract*)

Dr. H. B. Mulholland, University, Va.—Since James Hope in 1833 first used the term cardiac asthma to describe a syndrome occurring in patients with heart disease, the condition has been generally recognized and reported by various workers in this field. The similarity of many of these attacks, which occur in the middle of the night, to bronchial asthma, is most striking. Osler was one of the first to call attention to this. Also the precipitating factors found in many of these cases, such as emotional disturbance, fatigue and various physical upsets are known to bring on an attack of bronchial asthma. The association of wheezing with attacks of paroxysmal dyspnea has not been satisfactorily explained. The fact that cardiac patients may often have paroxysmal dyspnea without wheezing should be noted.

Cardiologists, though thoroughly recognizing the condition, have not been agreed on the pathological physiology giving rise to this train of symptoms and signs, nor is there any common agreement in the methods of therapy to be used during an attack.

I must confess that I was rather skeptical regarding the association of asthma and heart disease when Dr. Swineford first told me that he was embarking on this investigation and his findings were a very distinct surprise to me. The large number of patients suffering from heart disease and paroxysmal dyspnea accompanied by wheezing, who gave a previous history of definite and important allergic manifestations, in this paper is most striking and the conclusions arrived at seem to be logical. To the internist or cardiologist treating heart disease these facts should be of importance and it is probable that by a more thorough cooperation with the allergist in these cases some benefit to the patient may result. In other words, a careful examination by a competent allergist may result in more complete control of these patients and less frequent attacks of cardiac asthma.

It should also be emphasized that the precipitating factor in the attack of asthma in most instances is heart failure and, therefore, that this must be recognized as the fundamental condition and so treated. I thoroughly agree with Dr. Swineford that the term cardiac asthma should be reserved for those patients exhibiting paroxysmal dyspnea associated with wheezing.

Dr. Benj. M. Baker, Jr., Baltimore, Md.—Dr. Swineford's observations throw an entirely new light upon a most interesting topic. Cardiac asthma, also frequently called paroxysmal dyspnea, is a subject about which authorities have long confessed a certain confusion. During recent years, however, it has become quite widely accepted that paroxysmal dyspnea is a phenomenon of sudden failure of the left ventricle of the heart. Clinicians have long been familiar with the fact that apparently under similar circumstances these paroxysmal attacks of dyspnea occur in different forms. A patient with no auscultory abnormality, on examination of the lungs simply pants for breath. Another patient will present such wheezing in association with breathlessness that the picture simulates almost precisely attacks of non-cardiac bronchial asthma. And finally a third patient presents the classical manifestations of acute pulmonary edema.

Dr. Swineford's observations very strongly suggest that the patient who wheezes with his attack of paroxysmal dyspnea has wheezed before he developed cardiac disease. It does not seem unreasonable to me to assume that these patients' bronchi, by their previous experience, have become conditioned to ready spasm. The mechanism responsible for this formerly depended upon an allergic state. With the development of left ventricle failure acute pulmonary congestion may be sufficient bronchial irritant again to cause spasm and wheezing.

I should like to emphasize a further point. New observations of the sort Dr. Swineford has reported often misdirect us. I am sure that from the point of view of therapy he does not intend to do this. Cardiac asthma, whatever its precise mechanism, is fundamentally a phenomenon of grave failure of the heart. And, therefore, therapy must conform primarily to those principles which long and established experience has taught are most likely to aid the failing heart.

Dr. Swineford (closing).—We hope that this will point the way for detailed studies leading to a more satisfactory knowledge of the vexed problem of asthma and heart disease, particularly so-called cardiac asthma.

CARDIOLOGIC SIGNIFICANCE OF EPIGASTRIC PAIN*

By CAMP STANLEY, M.D., B.S., F.A.C.S.
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In view of the definitely marked and steadily progressive increase in deaths from cardiac disease in this country, it would appear that there should be closer cooperation between the gastroenterologist and the general internist in the prompt and correct correlation of clinical and subjective symptoms involved in generalized and thus frequently ill defined epigastric distress.

All physicians are well aware of the fact that epigastric pain is associated indirectly with a wide variety of thoracic and abdominal conditions which occur in their daily practice. For example, to cite only a few of these conditions, gastric and duodenal ulcer, acute pancreatitis, gallbladder disease, pneumonia, coronary occlusion and the various degrees of coronary thrombosis, pleuritis, and diaphragmatic affections are

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all included among the commoner causes of epigastric pain.

At this point a brief descriptive review of the nerve supply to the abdominal region will perhaps not be out of place. The abdominal walls, front and sides, are supplied by the lower six intercostal spinal nerves, both anterior and posterior branches, and terminate in the recti muscles on each side, conveying sensation to the midfront of the abdomen, while the posterior wall is supplied by the lumbar and sacral spinal nerves, all being intimately connected with the sacral and lumbar plexus, the pneumogastrics on the esophageal wall, the right to the posterior and the left to the anterior wall of the stomach, both entering the formations of the solar plexus, while the sympathetic enters the abdomen via the pillars of the diaphragm and the three splanchnics, which also enter the abdomen from the thorax.

From even a cursory examination of the intricate innervation which is here involved, there can be little wonder that epigastric pain may acquire widely varying degrees of significance. However, since we are here concerned only with the epigastric region, we have to consider chiefly the solar plexus and the nerve contacts of the upper abdomen.

Of all the pathological conditions enumerated above, there is one which is assuming an ever increasingly important position in medicine and is at the same time frequently associated with upper epigastric pain, namely: coronary occlusion and thrombosis. At the outset of an attack, while the pain is intense and of a more or less general character, it is practically impossible to confirm its cardiac origin. In many instances a cardiac seizure is preceded by an attack of apparent indigestion with nausea and vomiting, followed after a lapse of time varying from a few minutes to several hours by the onset of the acute epigastric pain of which the patient complains. While the subjective symptoms presented within the first few hours following an attack render the diagnosis difficult, after the expiration of twenty-four hours the differentiation of coronary occlusion and thrombosis from biliary disease, perforated ulcer, renal colic, and pneumonia becomes a comparatively simple matter.

The numerous deaths from so-called "acute indigestion" reported almost daily in the public press unquestionably result from coronary disease aggravated by the physical effort of patients to empty the stomach of a poorly selected combination of foods. Often in these instances the attempt to relieve initial epigastric pain by the introduction of comparatively heavy doses of bicarbonate of soda into an already overloaded stomach, added to the mechanical swallowing of air, so distends the stomach that the distress is frequently aggravated by actual mechanical pressure on the diaphragm.

Within the last few years an increasing number of cases, some of them involving patients with gallbladder disease, and some patients suspected of suffering from ulcer, have been referred to me. Practically without exception, these patients had been relieved of their original distress by their medical attendant. Only with the time required for a more careful detailed study of each individual case was the diagnosis finally definitely associated with coronary disease. The following case will serve as an illustration:

In February, 1933, I was called to see a man of 55 years, who complained of persistent epigastric pain which had gradually increased in severity for three days, during which there had been two attacks of nausea and vomiting. Two days later he was seized with acute dyspnea without marked increase in epigastric pain. At this time it was quite apparent that the latter was due to coronary disease, the existence of which was almost immediately confirmed by the cardiological study. Seven months later this patient developed an agranulocytosis with a fatal termination.

In the ordinary cases of angina pectoris with which we are familiar, the pain is substernal and radiates to the epigastrium, but in atypical cases the very early manifestations occur high up in the epigastrium and are readily confused with minor gastric disturbances.

For example, a middle-aged headmaster of a school for boys was seized with an attack of violent epigastric pain, followed six months later by a similar acute attack, both relieved by massive doses of morphine. Despite the fact that all laboratory and roentgen findings were negative, a diagnosis of gallstone colic was made. There was only one symptom, namely, intense pain in the epigastrium, with some evidence of dyspnea following the second attack. Because of the gastric symptom, perforated ulcer and acute pancreatitis were considered as possibilities, on account of the absence of any distention and the presence of a relaxed abdomen. I felt that an atypical coronary thrombosis was involved. Within seventy-two hours after my examination of the patient there was definite evidence of cardiac insufficiency. The diagnosis of thrombosis was subsequently confirmed by the cardiologist, and the electrocardiogram revealed a clearly defined myocardial infarct. With rest and the administration of the proper cardiac drugs the myocardial insufficiency and the anginal attacks subsided. The patient now enjoys a moderate degree of good health.

Epigastric pain is not at all uncommon in pneumonia of a lower lobe, pericarditis, or pleuritic involvement. No hypothesis based upon the mechanism of reflex radiation of pain would

explain why the reference of thoracic pain to the epigastrium should occur in one individual rather than in another, unless as a result of immaturity of nervous development of the individual. It is believed to be a generally accepted fact that in those cases of extra-abdominal disease (such as a pathologic pharynx) which produce epigastric pain, the sensory impulses via the medulla reach the vagus and glossopharyngeal nerve, and thence are conveyed to the gastro-intestinal tract.

Within the brief period of time at my disposal I have endeavored to describe the common experience of the gastro-enterologist with the particular type of epigastric pain which indicates the presence of cardiac disease. Here the gastro-enterologist is afforded an opportunity to co-operate with the cardiologist by referring patients suspected of suffering from diseases of the heart to the latter for the proper care.

DISCUSSION (*Abstract*)

Dr. Ernest H. Gaither, Baltimore, Md.—For many years I have been deeply interested in the endeavor to solve the many problems presented under the guise of epigastric pain. About sixty different disease entities may produce pain and discomfort in this area.

A discussion of the nerve supply and its mechanism in relation to symptomatology in this region is definitely indicated; however, time does not permit of such a presentation.

It is a well-established fact that in not a few instances, cardiac disease in the form of angina, coronary disease and myocardial degeneration gives the first evidence of its presence in persistent dyspeptic symptoms, namely, nausea, epigastric fullness and distress, lack of appetite and gaseous eructation; and at this stage, if there are no other manifestations, it is usually quite impossible to make a definite diagnosis of cardiac disease. However, if the symptoms described do appear in an individual past middle life presenting arteriosclerosis, hypertension and a history of having been engaged in laborious pursuits, one should at least have his suspicions aroused and thus be on the alert for any other indications such as fatigue, dyspnea, slight edema of the ankles, and cyanosis, which, though slight, may on different occasions appear.

One should minutely question patients with epigastric pain as to whether they suffer with dyspnea upon exertion, pain down the arm, or constriction through the chest necessitating immediate rest before relief is obtained; also whether epigastric pain is inaugurated by exercise and relieved by rest; or if dyspnea is an accompanying symptom; and if the replies be in the affirmative, our suspicions of primary cardiac disease, such as angina, or coronary or myocardial involvement, should be aroused.

There is one point I always endeavor to clarify; that is, whether the pain in the epigastrium is brought about or intensified by bodily exertion following a meal. This, to me, has always been a very significant and indicative symptom of heart involvement.

Not a few cases of coronary thrombosis have been operated upon because of the mistaken diagnosis of perforated ulcer, gallbladder or pancreatic disease, or acute intestinal obstruction. It therefore behooves us all to be upon our guard in every case in order to avoid such an untoward happening. It is well also to bear in mind that fever and leukocytosis may be present in coronary occlusion, and these clinical changes may serve to mislead us as to the possibility of cardiac disease.

One dare not leave this important subject without comment upon a group of cases presenting true cardiac disease originating in disease of the liver and gallbladder, particularly the latter. It is to be recalled that this very notable disease entity was first pointed out by T. R. Brown. In such cases there is every evidence of true organic heart disease. However, the symptoms in whole or in part disappear after the removal of the offending organ, that is, the gallbladder; phenomenal recovery ensues and the patient is transformed from a confirmed invalid into a healthy and useful citizen. To be sure of one's diagnosis is no easy task, I can assure you, and it calls for great moral courage in some instances to insist upon operation in the presence of few or no sustaining diagnostic facts, more especially when gallbladder visualization tests have proven negative. In such cases we must meticulously study the history as regards the onset of attacks; jaundice, slight or marked; chills, fever, changes in the color of the stools and urine, and radiation of pain; in short, every scintilla of evidence available which will be helpful in justifying a diagnosis of primary hepato-biliary disease.

While coronary thrombosis may simulate acute catastrophes, such as perforated ulcer, acute pancreatitis, cholelithiasis and acute intestinal obstruction, yet such a state does not reproduce an accurate picture of chronic ulcer.

It is of the greatest importance to emphasize the fact that individuals in the fourth and fifth decades, who present sclerotic changes and are suffering with true cardiovascular disease, may, and often do, also harbor hepato-biliary disease.

The problem under discussion is a momentous one, and in all cases presenting epigastric pain, particular consideration should be given to the possibility of cardiovascular disease.

Dr. James Alexander Lyon, Washington, D. C.—Dr. Stanley has brought out a point which needs to be emphasized repeatedly; that is, that certain cardiovascular conditions may at times give rise to symptoms which are predominantly epigastric.

Why coronary disease produces gastro-intestinal manifestations is obscure, but that such a relationship exists there is no doubt. We know, for instance, that in the early stages of angina pectoris the pain in some cases occurs only when exercise is taken following a hearty meal. The patient, unaware that exertion was the actual precipitating factor, relates his distress to overeating and consults a gastro-enterologist. I have recently had two patients with angina pectoris who had been treated for gastro-intestinal ailments, one for two years and the other for over a year, before the anginal nature of their attacks was suspected.

In some of these atypical cases the clue to a correct diagnosis may be found when a careful questioning has elicited a history of exertion preceding the onset of pain. Another clue may lie in the patient's tendency to keep

an upright position and to remain immobile during an attack of angina pectoris. The effect of a nitrite upon the pain may also furnish evidence as to the nature of the pain.

As Dr. Stanley has pointed out, in cases of coronary occlusion which closely simulate gastro-intestinal diseases there may be no clear differentiating sign or symptom present during the first twenty-four hours.

It should be remembered, conversely, that when a gastro-intestinal upset occurs in a patient who has arteriosclerotic or hypertensive heart disease, a cardiac neurosis or a labile nervous system, cardiovasular symptoms may predominate. I should like to cite an example. I was called one night several years ago to see a man in his early seventies, an eminent jurist, who was in extreme shock. His skin was cold, clammy and ashen gray, with cyanosis of the mucous membranes of the lips. He was suffering intense, continuous epigastric pain radiating into the chest and shoulders. There was moderate dyspnea, a fall in blood pressure and a weak radial pulse. The abdomen was markedly distended and tympanitic. In reply to questions his wife stated that earlier in the evening he had taken one or two cocktails and eaten chilli con carne and other indigestible foods. In this instance it would have been easy to confuse coronary thrombosis with the attack of pylorospasm from which the patient was suffering and to have treated him over a prolonged period for coronary infarction. The symptoms quickly subsided after the removal of the undigested food.

In these days when coronary disease appears to be on the increase in the age group of thirty and over and when its importance is rightly being recognized and stressed, we should not allow ourselves to swing to the other extreme and see coronary thrombosis and angina pectoris in every acute gastro-intestinal upset which occurs in a cardiovascular patient.

SOME REMARKS ON THE TREATMENT OF ALCOHOLISM*

By ERWIN WEXBERG, M.D.†
New Orleans, Louisiana

A man, 32 years of age, who came to see me some months ago, with his father, complained of serious nervous trouble which he himself reported to be due to excessive drinking for the previous five or six years. His history was the usual one for alcoholics. Being a football coach by profession, he had drunk very little as long as he was himself active in his sport. Later on he liked to have a social drink or two and he did not mind getting drunk once in a while. Finally, he got the habit of drinking every day, even when he was alone. The more he drank, the less he enjoyed it; however, when he made an effort to stop drinking he suffered from severe agrypnia and intolerable restlessness, so that he started drinking again. None of his numerous attempts at curing himself had lasted more than two or three days. During the preceding weeks he felt very bad. Before going to sleep he had hallucinations, seeing little animals crawling around. When he finally fell asleep he waked from terrific nightmares and passed hours in a state of agonizing anxiety, until he put himself to sleep again with a considerable amount of whiskey. In the morning he waked up nauseated and vomiting, and felt better only after another drink.

Neurological examination of the patient revealed nothing of importance, except a rapid, coarse finger tremor, typical of alcoholics. His state of mind was one of apprehensive restlessness; however, there still was no symptom of psychosis. He was well oriented as to time, space, and personal affairs. His recent memory was fairly good. No delusions or hallucinations were present at the time.

Considering the patient's report of typical hallucinations and his state of apprehensive excitement, I felt justified in considering this as a case of alcoholism on the verge of delirium tremens. Permanent sleeplessness, only to be conquered by increasing amounts of alcohol is a usual feature in the premonitory stage of alcoholic delirium. For important reasons, commitment to a mental hospital was out of the question. The patient did not have funds enough to enter a private institution, and he positively refused to seek admission to a state hospital. Therefore, I decided to make an attempt to treat him in his own home.

I started by frankly telling him about his condition, pointing out to him that he would have to be committed, even against his will, in a few days, in a state of delirium, if things went on as they were. I wanted to frighten him, and I did so, in order to secure his full cooperation. Then I put him to bed and ordered high vitamin diet, including fruit, fruit juice, tomatoes, green vegetables, and brewer's yeast tablets. After a blood sugar test had been done, which turned out normal, I started injections of insulin, giving five units once a day. At night I ordered 1.5 grains of sodium phenobarbital.

The result of this treatment was much better than I had expected. From the first day the patient was much quieter than before, and after one week nervousness had subsided altogether. At that time phenobarbital was discontinued and the patient slept the whole night as he had not slept for years. His appetite increased, and he gained slightly in weight. After two weeks insulin was discontinued, and now, six months since the beginning of treatment, the patient is feeling fine, entirely abstinent, and still living on high vitamin diet.

Here my report is finished, as far as physical treatment is concerned. However, we are aware of the fact that chronic alcoholism is only secondarily a physical condition, and primarily a mental one, and that it scarcely makes sense to get the patient over the physical predicament of abstinence without taking care of the underlying mental condition. Therefore, I started a series of psychiatric interviews as soon as the patient was well enough and able to concentrate. As for his psychiatric history, it was, shortly, the following: he was the only son of a successful self-made man, and was considerably spoiled as a child. There was no trouble during his childhood and youth, his parents were ready to

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give him all the pleasure he might desire, and life ran smoothly and without conflict. He was fairly successful in sport, and so it was easy for him to make sport his profession and to become a coach. At 23 years of age he married. Sex life was unsatisfactory, the wife had no understanding of her husband's easygoing way of living. Within five years three children were born. The wife kept her unconcerned and stern attitude toward her children and husband, and the patient took to drinking more and more, since it meant some compensation for what he felt was missing in his life. During the frequent quarrels he had with his wife, he found out that his drinking was the one thing that hurt her more than anything else, and he got some satisfaction from that. Three years before I saw him they separated. At that time the patient found himself already entirely unable to cope with his habit of drinking, even with some good will. One year and a half before treatment he lost his job, and then things went from bad to worse. His parents could not hide their disapproval. They also nagged and criticized him, as his wife had done before.

The patient was made to understand his own responsibility in the development of his condition. He had been able to get along as long as things went on by themselves. He failed when difficulties arose and when it was up to him to do something about them. Keeping the attitude of a spoiled child during his grown-up years, he was not prepared to take any frustration, and so he chose drinking as a substitute for unearned and unpaid for satisfaction he had enjoyed in his parents' home.

A free discussion of his problems helped him to find himself again. He decided to ask his wife for a divorce and to arrange his affairs in a way suitable to a new start. He secured work.

Of course, time is too short to consider this patient as cured. We should not be surprised at all if he had a relapse one of these days. However, in this case as in others that I have seen, it was possible to break the habit of chronic alcoholism without violent methods or restraint, because the patient's cooperation was secured. This holds to a certain extent for drug addicts also.

I ordered a high vitamin diet in accordance with experiences that I had with that diet in a series of cases of alcoholic psychoses, as well as polyneuritides and spinal involvements.*

*See Wexberg, "Diseases of the Nervous System from Nutritional Deficiency." Southern Medical Journal, March, 1937. Further literature quoted there.

The recent conception of alcoholic polyneuritis as due to avitaminosis ought to be applied, practically, to alcoholic psychoses. In fact, I have seen delirium and hallucinations clear up in an unusually short time with that treatment. Whether the therapeutic test in these cases suffices to prove that alcoholic psychoses are in part or entirely due to metabolic disturbance from avitaminosis, I should rather not decide, nor even discuss on this occasion. Certainly there might be more than one cause to account for nutritional deficiency in alcoholics. There is the fact, for example, that they usually do not care for food, or that gastro-intestinal disturbance and deficiency of absorption exist in almost every case, or that relative vitamin deficiency may arise from caloric surplus in an alcoholic's nutrition. However, it is possible that an increased vitamin supply may have some pharmacological effect even if the condition concerned is not due to deficiency at all.

As a practical procedure, increasing the patient's vitamin supply can be recommended as a routine treatment in alcoholic psychoses. I believe that the fact that our patient was able to stop drinking without any abstinence symptoms whatever was mostly due to the dietary regime followed.

The administration of small doses of insulin which I have found useful in the treatment of alcoholism, reminds us of the recent reports about insulin shock therapy in schizophrenia, which was started by Sakel in Vienna and which now is in the stage of trial in this country. While I am unable to say anything about this new treatment because of lack of personal experience with it, I want to say that the use of small doses of insulin, which was recommended for the treatment of alcoholics and drug addicts several years ago, probably has nothing to do with insulin shock therapy. The most probable explanation of the former seems to be the one which claims that the sensation of intense hunger which is brought on by insulin makes the patient care for food rather than for drinking. This looks like a psychological theory, but it is not; for the condition of an individual's craving for food is primarily physical, though naturally it has its psychological implications. It is on the same line as the sometimes successful insulinization of patients suffering from gastric crises in tabes, the vegetative condition of intense hunger being strictly

antagonistic to that of nausea and vomiting typical of gastric crisis.*

However, while giving full consideration to these physical factors in the treatment of alcoholism, undoubtedly the psychiatric approach is the more important one. Whenever real cooperation from the patient's side cannot be produced, no permanent result from any method is to be expected. Therefore, the prognosis of the single case depends entirely on the patient's personality makeup and his situation in life. In a very interesting book recently published ("The Exploration of the Inner Mind"), the author, Boisen, established the fact that recovery in schizophrenia largely depends on the patient's premorbid personality and life situation. In other words, those patients who were primarily "good material" and who have something to look forward to, in their own lives, a family, a good job, and so on, are more likely to recover than others. What seems to hold for schizophrenia, notwithstanding its organic background, is quite obvious for alcoholism. Much can be done for alcoholics and drug addicts by increasing social security in general; still more, I believe, by a psychiatric treatment which gives them another outlook on life and enables them to meet difficulties with better mental and emotional equipment than they had before. Special institutions organized for that purpose are an increasing necessity.

*Chen, Ch'eng and Lyman, in a recent paper on Insulin Treatment in Morphine Addiction (Jour. Nerv. & Ment. Dis., 83:281, 1936), have the same explanation in mind for the good results in their cases.

MENTAL HYGIENE IN CHANGING TIMES*

By W. W. YOUNG, M.D.†
Atlanta, Georgia

Mental hygiene gained new impetus and the present day trend had its beginnings with the recovery of Clifford Beers from a mental illness. Having been a patient in mental institutions where he suffered many indignities because of the ignorance of an archaic system, he gave out a chronicle of conditions all too prevalent in institutions for mental disease. These conditions cried aloud for reform. "The Mind That Found Itself" resulted in the organization of the National Committee for Mental Hygiene for this purpose.

An investigation was undertaken on a nation-wide basis. This soon led to a definite shift in philosophy from custodial care in "insane asylums" to treatment in "hospitals for mental disease." No longer were men and women to be placed in institutions simply because they could not be tolerated by the family or the community. They were to be placed in hospitals for treatment in order to get them well and return them to the community as effectual citizens.

The stress on treatment of necessity must lead to a study of causation. Etiology after all is the backbone of all treatment. More intensive work along research lives evolved from this. In these studies of causation the trail of the disorder led back over and over again to childhood. For this reason the stress of studies began to be laid more and more at this point. For this purpose experimental "Child Guidance Clinics" were set up throughout the country so that intensive studies of disorders in their incipiency might be made. A report after ten years by Dr. George Stephenson reveals the tremendous importance of this movement. Such study showed the major role played by the parent-child relationship and the fact that much can be done through parent education and guidance toward prevention of growth into maladjustment. Consequently, one of the most important factors in a mental hygiene program rightly has come to be that of prevention.

Out of this study has evolved a definite mental hygiene philosophy. This philosophy teaches that human problems should be dealt with, not on the basis of overt behavior alone, but more particularly on the basis of the motivations of this behavior. The concern must be shifted from the fact that a theft has been committed to a study of the reasons for the theft; from the fact that two nations are at war, to a consideration of why this has taken place. This philosophy concerns not only the individual as related to his own inner problems but also all human relations: the husband-wife relationship, employer-employe, legal relations of judge and community to the criminal, teacher to child, and all such points of human and emotional interaction.

During these years psychiatric education has increased markedly in efficiency. Twenty odd years ago the course in psychiatry was something tacked on to the curriculum to give the student a little knowledge of the classification and nomenclature of the major mental disorders. It usually consisted of sixteen or more hours of

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didactic work or, if there was a clinical demonstration, to the student it was more of the nature of a glorified side show. Students looked upon the whole subject as unnecessary because it dealt with a specialty with which they never expected to have contact. Today psychiatry is playing a major role in medical education. Courses usually run from three to four years. The purpose is not to teach the student psychiatry *per se* but to instill the mental hygiene philosophy so that he will deal with patients as persons who incidentally possess diseased organs, and with himself in his human relations in general on a more understanding basis. The average physician graduating in recent years should thus be better equipped to become a more effectual member of his profession and his community.

During these years in which the mental hygiene program has been evolving, industry has developed with almost mushroom growth. The nation has become more and more mechanized. This is truly a machine age, and with the growth of factories with production as a slogan and the constant invention and use of labor-saving machinery, things have been happening to our populations too numerous and too subtle to be mentioned in detail. Life has changed from being predominantly rural to predominantly urban. Practically everything used by a city dwelling population is manufactured ready-made for its use.

These things have had a definite effect upon the home. The home used to be a place of activities where clothes were made; canning, pickling, preserving, and a multiplicity of duties performed. Everyone was busy; each one had his chores. Today it is much easier and often cheaper to buy pickles, preserves and jellies at the grocers. Better and cheaper dresses may be bought at the department store. Whole meals may be picked up by the housewife returning late from a bridge game. There is no incentive to activities and the home is no longer a center for them. Consequently, it has ceased to be a place to go to and has become a place to go from. Children and adults both, but more particularly the former, are apt to have too much purposeless unoccupied time, often the opportunity for learning bad habits and drifting into antisocial behavior. The home as an institution is in a state of flux out of which something stable must evolve.

The multiplication of machinery has made the necessity of employment of labor increasingly less. This has brought in its wake necessary unemployment due on one hand to high specialization and on the other to the vicissitudes of industry. As a consequence, there is more enforced leisure. "The idle mind is the devil's workshop," and again in this culture antisocial behavior is often bred. No methods have been evolved to keep the social structure in pace with the development of industry. Our social structure, in relation to industry, reminds one of a fast-growing ten-year-old who has never discarded his five-year-old pants. Women have been "emancipated" and now occupy every position in life. Formerly a woman could either teach school or marry or be that most terrible of creatures, an "old maid." She was either good or bad. These were nice easy rules to play the game by. Today the old standards are outgrown and ditched and no new and adequate ones have been evolved to take their place. This leads to insecurity and confusion. Children see nothing in continuity. Cause and effect, the evolving through process into something, is not apparent. Pork comes from the butcher, butter from a grocer, clothes from the department store. They fail to see their place in life and what its relationships are. This leads to confusion. Panic tends to come to men with the uncertainties of employment.

Civilization has evolved certain institutions which are supposed to guide, direct and care for human beings in the vicissitudes of life. Government, the courts, the church, the schools are supposed to play this role in our social structure. They should be the dominant factors in keeping this social structure in working order. They have failed. Years ago a man in New York City was pleading for free education for the masses, for public schools. He promised that, should such schools be founded, in less than a century our jails and insane asylums would close their doors. Education is now accessible to the large majority of people in this country. Law enforcement machinery has become more and more intricate and a greater and greater burden upon the taxpayer. Laws have been multiplied *ad nauseam*. Churches have grown in numbers. Yet today we are faced with the fact that, instead of a decrease of all types of social and personal maladjustments coming under the heads of crime, insanity, and chronic dependency, there has been a steady increase. There can be only one answer: our institutions have failed of their promise.

This must call for a change in method, a relinquishing of old, wornout philosophies. Pragmatically there is no other answer. They should

all take on a mental hygiene philosophy. The courts should stop meting out punishment on the basis of overt acts on an arbitrary graduated scale but should handle individuals as such with an effort to understand motivations and handle accordingly. The legal profession should cease to talk in terms of precedent and take on more vision. The minister should cease to inveigh against acts as his major duty and get a more personal touch with more understanding of their motivations. The schools should think more in terms of emotional adjustments and less in terms of academic education.

Faced with the threat of failing structures people find themselves in a state of insecurity at all points. The home has changed, whether for good or bad no one knows. Our institutions in which implicit faith for guidance and preservation has been placed, have failed to keep the social structure in pace with industry. Employment is uncertain. The very seeds of escape into neuroticism or something worse are sown in the air. Our institutions must be revamped. Too long have reactionary tactics based on precedent been dominant in government. They have failed. Men of vision who will do some cautious, judicious and thoughtful experimentation along new lines based upon a mental hygiene philosophy are needed. For after all, life is an experiment, but in which intelligence and pragmatism should guide. Change in government policy should include the enactment of social security legislation so that man in this new era may be insured against the vicissitudes of life to lend him that modicum of security, his right in these changing times.

The mental hygiene movement then must enlarge its scope to take in education of the people in its philosophies to the end that more pragmatically sound institutions may evolve and more consequent security be engendered into the mass of mankind. When every institution making up our social structure has taken on this philosophy; when every policeman, judge, minister, physician, school teacher, and parent, begins to approach human relations from this viewpoint, then the mental hygiene program may be said to be finished. This should be its goal: to work itself out of existence.

DISCUSSION (*Abstract*)

Dr. Esther L. Richards, Baltimore, Md.—This paper puts into plain, concise statements what each and everyone of us has been feeling for a long time, and that is the fact that our concepts of mental hygiene as a movement have long since outgrown the swaddling clothes of twenty-five years ago. These swaddling clothes consisted of certain beliefs that seem to make little allowance for the great fundamental principles of human behavior that lie at the root of well-balanced living. First of all, there was the idea that from a greater abundance of formal educational opportunities there would inevitably arise a higher degree of emotional maturity; that in giving individuals a greater opportunity to grasp intellectual insight into their selfishness and egotism and petty strivings and fundamental jealousies, they would overcome these Adamic characteristics. The fallacy in this line of reasoning was due to our failure to realize that there are two great principles involved in something we call insight. One is the intellectual ability to grasp a point of view, and the other is the individual's emotional willingness to accept what his intelligence presents to him. The general principles toward making higher education the goal of youth have resulted in a great demand for academic tickets of degrees. But education has made only a very feeble start in a program which takes any serious thought of the human material which is getting the degrees and the diplomas. We have only to look at the spots in college and university curricula that are drawing increased enrollments, such as banking, industry, economics, to see that the trend of interest in youth lies toward the commercialization of academic interest and away from subject matter which contributes toward a really broad concept of personality education opening up to the student the wealth of human experience bound up in history, literature, art and philosophy. Certainly raising the academic standards of requirements for vocational functioning by no means insures a real degree in education.

A second disillusioning belief was that raising standards of living must necessarily raise standards of life. We have only to look at the chaos within our midst to see the trends which standards of living have taken and the interpretations which have been put upon something called standards of life. Never before in the history of this country have we witnessed such a spectacle of class distinction as has passed before us in the political campaigning of the last six months. I need not go into its gruesomeness.

A third disillusioning belief was that which arose within the mental hygiene movement itself. It was the propaganda of statement that led its hearers to believe that could we get at the child early enough in the home and in the school we would reduce our state hospital and penitentiary population. Here again was the idea that one had only to bring high-powered pressure of intensive psychiatric social work to bear upon parents and teachers and our parent education problem would be solved. The increase in crime and delinquency and psychiatric hospital patients in the presence of magnificant child guidance budgets took the bottom out of the child guidance movement as it originally started. We have learned the reason, I hope, that reform and reeducation are not procedures that can come from without, but an achievement that to be

wholesome must come from within. Only in so far as the home and the school and the social set and the community comes to realize its need for help can real mental hygiene service be accomplished. Years ago when Mr. Beers conceived the idea of a mental hygiene educational activity his first thought was to begin with legislature. It was Dr. Adolf Meyer in those far-away days who urged him to abandon that form of campaigning and to start with surveys designed to get some knowledge of the mental health needs of communities, of schools and homes, and public and private welfare projects; to start in a modest way in one spot not in advertising and prophesying what should be done, but studying quietly what could be done with parents and teachers in the slow, day-by-day, undramatic experience of human contacts. There is such a thing as over-organization and over-campaigning in any experience of human welfare. Small beginnings, if rightly started, grow into large educational experiences, and no movement can grow faster than the capacities of its individual and group components. The machine in industrialism, the radio, the apartment house, the moving picture, the airplane in our community life are facts of evolution that are bound to come, and are not in themselves causes of social disorganization except in so far as they are made by the rigidities of traditional attitudes and living which man is loth to give up. Civilization's assimilation of these innovations comes either by panic and revolution or by gradual expansions of mental attitudes and traditions to meet changing times. It is a giving up and acceptance and finding new ways and means of living that constitutes assimilation. We are in changing times, and no amount of kicking and screaming will bring back conditions of the past. It is in such times as this that we are forced to pause at the crossways and realize that we are obliged to face such philosophies of living together that are not amenable to childish conflicts of political partisanship. Increasing dependence and poor mental health call for increasing community consciousness of responsibility and wise guidance for those who are handicapped. In our social thinking we are not confronted with tiding over something called a "depression," but we are facing the serious consideration of certain community and state and national responsibilities to be carried in the future by private organizations or by a more communally organized responsibility. The facts are before us and around us and engulfing us. As Dr. Young has said, the church, the law, the educational system have got to wake up to assuming an active and intelligent part in the settling of great problems that have to do with public health and social welfare. Mental hygiene must rise from the status of a scientific phrase and an extraneous reforming movement to a status of meaning something in the actual functioning of individual lives before it can become a great constructive force in our public welfare.

Dr. R. Finley Gayle, Jr., Richmond, Va.—There is nothing controversial in Dr. Young's paper, and I, therefore, cannot take issue with him on the matter he has presented.

There are several important points brought out by him which I should like to emphasize. We all realize that if we are to accomplish anything toward the prevention of mental disease, we must emphasize cause and not effect in the teaching of psychiatry. It is all too true that in the teaching of psychiatry everywhere, until quite recently, more emphasis was laid on the classification of mental disease than on what caused it. Undoubtedly a certain number of psychoneurotic and psychotic states in adolescence and adult life will recover under simple custodial care, and at times under no treatment at all; but how many individuals would have been spared these abnormal reactions which incapacitated them, had they received the proper mental guidance at home and in the school! How many unhappy domestic and vocational situations could be alleviated should these individuals so affected get adequate psychiatric help to learn why they are mentally sick and thus relieve the condition!

After all, it is a question of education, mental hygiene education, not only education of the physician, but education of the parent and school teacher; and as Dr. Young says, "When every policeman, judge, minister, doctor, school teacher, parent, etc.," accepts the point of view of the mental hygiene program, then we have finished our labor. The growth of the mental hygiene movement has been undoubtedly retarded by the economic depression which we have recently passed through and all of the great foundations had of necessity to curtail their activities and thus lessen the momentum of this movement and much ground was lost. Education along the lines of mental hygiene is absolutely essential so that everyone having to do with human behavior will have an insight into the life problems of those individuals they come in contact with, and will be able, because of this knowledge, to give them helpful advice or properly direct them where they may be helped.

Education of everyone having to do with human behavior can best be planned, guided and directed by properly organized mental hygiene societies. The South is woefully weak in this respect and there are few state societies effectively functioning in our territory. Some of our northern and western neighbors have done better. My plea would be that we all stress this education by organizing and promoting such societies.

Dr. Erwin Wexberg, New Orleans, La.—In spite of the fact that I heartily agree with all of what Dr. Young said about the importance of the modern mental hygiene movement, I do not believe it is superfluous to warn against a certain kind of mental hygiene propaganda which consists of improper popularization of psychiatric terms and conceptions. Since it is rarely possible to give the laymen all those explanations required to prevent misunderstanding, even the best intentions to be supposed being behind popular books on mental hygiene subjects may turn out badly and have disastrous effects. At times, even a title may do the same, as in the case of a recently published book named "Be Glad You Are a Neurotic!" You certainly will agree with me that it is not the right thing to encourage people to develop a neurosis and to be proud of it. I wish everybody who is interested in mental hygiene and wants to write popular books about it were conscious of the tremendous responsibility which is involved therein and would consider this more than his business interests.

SYMPOSIUM ON MALARIA—Part 1‡

THE FIRST TWELVE MONTHS OF INFANCY AS A TEST FOR THE COMMUNITY INCIDENCE OF INITIAL ATTACKS OF MALARIA*

By HERBERT C. CLARK, M.D.†
Panama, R. de P.

We are all aware of the difficulty one meets, in an endemic area, in trying to differentiate a new malarial infection from a recrudescence or relapse. It is our belief that the parasite rate we find each month in the native villages of the Chagres River Valley is very largely due to recrudescence or relapse. On the other hand, a year ago we had an epidemic in which we are reasonably certain that many new infections occurred because there were individuals attacked that had not, over a long period of time, shown parasites in their blood films on our monthly surveys. It seems that our only means of improving a *yardstick*, that can give us some idea of the chances and the time interval likely to be involved in acquiring an initial or a new infection when exposed to a life spent in such villages, is to study the babies that are born and raised there and to determine the age at which they approximate the parasite index for children. In the ordinary course of events, in a highly endemic area, what are the probabilities in the first twelve months of life in regard to initial infections of malaria and to recrudescence or relapse? All of us know that individuals, particularly of a non-tolerant race from a malaria-free region, have acquired malaria as a result of a single weekend spent in a bad area. We also know that the average person often spends a few weeks or months in such a region before malaria is acquired and, rarely, some individuals seem never to acquire the disease. Other features that demand consideration in the first twelve months of life are congenital or hereditary malaria, and the effect of the nursing period as a defense against the disease. I am inclined to doubt many of the reported cases of congenital malaria because the chance of contaminating the cord blood with the placental blood is very great, unless the blood films are taken by one fully competent to prepare them. The second reason for doubt is the time interval after birth that is recorded in many of them, which certainly permits the question of acquirement after birth. Cases of congenital malaria are extremely rare. I have seen only one in my 27 years of tropical experience and that resulted, I am sure, from surgical intervention in a case of placenta previa where the placenta was torn into the cord attachment.

Daleas[1] (1935) reports that Henry's reaction is often positive in the newly born children of Indo-China and he feels that congenital malaria is common though parasites are rarely found in the peripheral blood of the babies.

Wickramasuriya[2] reports six cases of congenital malaria that he considers the result of transplacental passage of parasites.

Schwetz and Peel[3] in a study of 56 women of Central Africa at the time of childbirth report that parasites were found in 6 per cent of the cord blood films and 3.6 per cent of the infant blood films.

Barbosa and Arjona[4] state that congenital malaria undoubtedly exists but that the percentage of cases is very small.

Blacklock and Gordon[5] find that the stagnation of blood in the placenta, with its limitation of oxygen, and the presence of glucose favor the localization and growth of parasites, but they did not find a case of congenital malaria in their study of 26 native women of Sierra Leone.

Wislocki[6] says, "It is unlikely that malarial organisms can enter or penetrate the chorionic syncytium, so that the transmission of malaria from mother to fetus occurs rarely, if at all.

Barber, Mandekos and Rice[7] have very recently reported on 1,575 examinations of infants one year of age or less in malarious villages of Greek Macedonia. In discussing the parasite index of infancy they make the following remarks:

> "It is theoretically possible that some inhibitory substance may be transferred from mothers to infants before birth or through lactation ..... The parasite index of 281 infants three or four months old was 45.6 per cent; that of 259 mothers was 59.1 per cent. If very young infants enjoy no natural protection against infection, it is difficult to see how a single infant of this locality could escape infection during an exposure of three weeks; yet we see that the majority of them escape for three months or more, so far as the blood examination could show. Whatever protection very young infants possess is not complete or universal. We have found two infants infected after 20 and 24 days of life, three after 4 to 5 weeks, and eighteen after 6 to 10 weeks."

My report[8] (1915) on the diagnostic value of the placental blood film in malaria and my continued experience since that date in Central America and Panama have not revealed to me a single case that I would list as congenital malaria save for the one already mentioned in connection with the surgical management of placenta previa. Nevertheless, I should present for your consideration some cases from my autopsy service at Ancon, C. Z. This service extended over a period of ten years and included a large part of the construction period of the Panama Canal when the negro families lived under very primitive conditions, yet I have but four cases of fatal malaria in early infancy to report and all of them are considered acquired (postnatal) infections:

Autopsy 2,866, age 3 weeks, West Indian Negro.
Autopsy 2,000, age 6 weeks, West Indian Negro.
Autopsy 3,756, age 2½ months, West Indian Negro.
Autopsy 3,805, age 8 months, West Indian Negro.

Even the first and second cases mentioned had the time and opportunity to acquire postnatal infections. Cases of congenital malaria are extremely rare, in my opinion, and when they do occur it indicates that some accident or disease has broken the placental barrier. Re-

*Chairman's Address, National Malaria Committee (Conference on Malaria), meeting conjointly with Southern Medical Association, Thirtieth Annual Meeting, Baltimore, Maryland, November 17-20, 1936.

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‡The Symposium on Malaria, papers and discussions from the National Malaria Committee, meeting conjointly with the Southern Medical Association at Baltimore, Maryland, November 17-20, 1936, which are to be published in the SOUTHERN MEDICAL JOURNAL, will appear in two parts: Part 1 in this issue, and Part 2 in September.

gardless of the massive infections one finds in the placental blood, the fetal blood is negative for parasites practically without exception.

It is, to me, astounding how few infants under two months of age are found positive for malaria parasites even when such babies are born and reared in camps and rural locations of high endemicity. Because of this, I am forced to wonder whether the mother and the nursing period provide these infants with some natural defensive mechanism and just how much reliance can be placed on the first two months of life as a yardstick for the incidence of initial attacks of malaria. In fact, the normal parasite index for children is not reached in our villages even at the end of twelve months although infections do become very numerous around the twelfth month. It has taken several years to build up a sufficiently large number of babies for analysis but it is possible now to present two groups. The first group[9] is made up of babies found in the Chagres villages and in various other rural parts of Panama before any control was instituted. These babies were not examined monthly for twelve consecutive months. Most of them were examined but once and the age recorded at the date of the survey (*Table 1*).

Table 1

BABY SURVEY

Various Rural Villages in Panama

Malaria Endemic, No Control Measures

Age in Months	Number Examined	Positive for Malaria
1 or less	6	0
1 to 2	7	0
2 to 3	13	3
3 to 4	16	3
4 to 5	7	0
5 to 6	11	5
6 to 7	17	5
7 to 8	16	2
8 to 9	9	6
9 to 10	11	0
10 to 11	12	6
11 to 12	95	20
Total	220	50

The second group consists of babies in our river villages that we were able to survey by the thick blood film method[10] on twelve consecutive months from the time of birth (*Table 2*).

Table 2

BABY SURVEY

120 Examined For The Parasites of Malaria

On 12 Consecutive Months From Birth

Mid-month Examination	Number Positive
First month	0
Second	0
Third	4
Fourth	1
Fifth	1
Sixth	0
Seventh	2
Eight	4
Ninth	4
Tenth	1
Eleventh	2
Twelfth	13
Total	32

During the first six months of life there were 60 examined and 11 found positive, a parasite index of 18.3 per cent.

During the second six months there were 160 examined and 39 found positive, a parasite index of 24.4 per cent.

A total of 50 positives out of 220 examined gives a rate for the twelve months of 22.7 per cent and this is about half what the children's index is expected to be for such places.

In addition to the 32 initial infections recorded there were 3 showing recrudescence, relapse or a new infection as the case may be. The first of these cases had its initial infection during the third month (*P.vivax*) and in the twelfth month revealed the parasites of *P.falciparum*. The second case acquired its initial attack in the fourth month (*P.falciparum*) and showed the same species of parasites in the fifth month. The third case revealed its first infection in the fifth month (*P.vivax* and *P.falciparum*) with the reappearance of one or the other of these parasites on the sixth, seventh and tenth months.

In addition to these two groups of babies, a third group of 66 has been in survey progress for the current year and they range in age from one to twelve months. It is not possible to compare these with the tables just shown since it will require 12 consecutive months' observation and some of them may not remain in the area or may not be present at all surveys. Thus far six of the number (9.1 per cent) have been positive for the parasites of malaria. Two of these had their initial attack in the seventh month, one in the eighth month, one in the ninth month, one in the tenth month and one in the twelfth month. Four of the cases had *P. falciparum* infections and two of them *P. vivax* infections.

DISCUSSION

These babies wear almost no clothing during the first year of life and are therefore exposed to mosquito bites more than other children and adults. They live near uncontrolled anopheline breeding areas and are not living in screened houses. Some of them spend the night hours under mosquito netting but the discipline in the use of the net is poor and one can usually catch more anopheline mosquitoes inside these nets in daytime than in other parts of the house.

In both tables there were no babies showing parasites until the third month. *Table 1* had a rate of 18.3 per cent for the first six months, *Table 2* a rate of 5 per cent for the first six months.

In the second six months, *Table 1* had a rate of 24.4 per cent, *Table 2* a rate of 21.6 per cent.

The total results in the two tables for the parasite index were 22.7 per cent, 26.6 per cent.

Table 2 shows a rate of 9.4 per cent of recrudescence, relapse or new infections for the babies of the Chagres River and the village of New San Juan as compared with a rate of 57.4 per cent among all others in the same areas for the same period of time and for twelve consecutive monthly surveys.

The annual cumulative parasite index for the current year in these same groups were 9.1 per cent (infants) and 58.2 per cent (all others above twelve months of age).

Perhaps the first two months of infant life are in some way provided a natural defense against the disease

but I feel that the remaining ten months of the first year offer us to some extent a helpful guide in regard to the frequency of new infections in an endemic region as compared to recrudescence and relapse. This disease is not so often eradicated by treatment and acquired tolerance as many of us think. The use of transfusions as a therapeutic measure in recent years has developed many surprises in the way of persistence of malarial infection. A malaria patient[11] following his recovery in the tropics spent the next nine years in Paris and was apparently well during this time but he then developed a large abscess and anemia. A transfusion was given him and his donor contracted malaria. The patient himself was then found to be positive for parasites and he had a slight relapse a few days later.

In another case, several ounces of an Italian's blood[12] were given as a transfusion to his anemic infant, which soon afterwards developed quartan malaria. Quartan parasites were then found in the father's blood. The father had no clinical symptoms of malaria but admitted that about once in 12 to 24 months he had a chill which lasted for an hour and was followed by perspiration. He had suffered from malaria as a child, but he had lived in Boston for the previous 20 years.

CONCLUSION

Study of the babies included in these three groups indicates that they can be used to some extent as a measure of new infections in an endemic area. It also shows that congenital malaria must be an extremely rare occurrence. The first two months of infant life appear to be protected either by something inherited from the mother or associated with the nursing period. Whatever this defense may be it is rapidly lost toward the close of the twelfth month.

REFERENCES

1. Daleas, P.: Malaria and Pregnancy. Bull. Soc. Med-Chirurg. Indochine, Vol. **13**:432-435 (May) 1935.
2. Wickramasuriya, G. A. W.: Some Observations on Malaria Occurring in Association with Pregnancy, with Special Reference to the Transplacental Passage of Parasites from the Maternal to the Foetal Circulation. Jl. Obst. and Gynaec. British Empire. **42**:816-834 (Oct.) 1935.
3. Schwetz, J. and Peel: Congenital Malaria and Placental Infections Amongst the Negroes of Central Africa. Trans. Roy. Soc. Trop. Med. & Hyg., **28**:167-174 (Aug. 4) 1934.
4. Barbosa, Amando; and Arjona, Benito Lopez: Malaria in the First Year of Life. Plasencia, C aceres: Imprenta "La Victoria" Valdegamas Numero 20, 1935.
5. Blacklock, B.; and Gordon, R. M.: Malaria Parasites in Placental Blood. Ann. Trop. Med. & Par., **19**:37-45 (March) 1925.
6. Wislocki, George B.: Observations on the Placenta from a Case of Malaria. Bull. Johns Hopkins Hospt., **47**:157-163 (Sept.) 1930.
7. Barber, M. A.; Mandekos, A., and Rice, J. B.: The Seasonal Incidence of Malaria Transmission in Macedonia. Amer. Jour. Hyg., **24**:258 (Sept.) 1936.
8. Clark, H. C.: The Diagnostic Value of the Placental Blood Film in Estivo-Autumnal Malaria. J. Exp. Med. **22**: (Oct.) 1915.
9. Clark, H. C.; and Komp, W. H. W.: Observations on Malaria Incidence In Some Unsanitated River Villages in the Republic of Panama, with Special Reference to Proposed Construction Projects in the Canal Zone. Sou. Med. Jour., **25**:642-647, 1932.
10. Barber, M. A.; and Komp, W. H. W.: Method of Preparing and Examining thick blood films for the Diagnosis of Malaria. Public Health Reports, **44**:2230-2341, 1929.
11. A Malarial Relapse after Nine Years. Transmission to the donor during Transfusion. The Danger of Transfusion in the Colonies. Rev. Med. et Hyg. Trop., **23**:32-37 (Jan.-Feb.) 1931.
12. Jankelson, I. R.: Transmission of Malaria Through Injection of Whole Blood. J.A.M.A., **97**:177 (July 18) 1931.

RECENT RESEARCH ON PROPHYLAXIS AND TREATMENT OF MALARIA*

REPORT FOR 1936

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The great volume of printed reports on these subjects since our last meeting makes it necessary to state that I have assumed the responsibility of selecting only a few of the representative reports from different countries for the use of our membership. For those who may want more details and additional reports I respectfully refer them to the League of Nations Malaria Commission and to the Tropical Diseases Bulletin. For the busy field worker I am sure the Bulletin will serve him well with its mass of concise abstracts covering all tropical and subtropical regions.

Quinine is still our drug for the masses and our yardstick to measure the value of other new drugs. Following the plan of last year's report, this review will be given to the new drugs that have received the most extensive field or hospital trials. The field workers in malaria control who are interested in having drug control added to their weapons in the anti-malarial fight will be interested in a recent statement by Watson[1] who says, "There is, in my opinion, no antagonism and no competition between the various methods of preventing malaria . . . . . in all campaigns antimalarial drugs have a place."

The Ceylon malaria epidemic has held our attention for some time. It is interesting to note that the medical authorities there always keep a reserve supply of quinine equal to ten months requirements and 7,000 pounds were available at the beginning of that epidemic. However, it became necessary to obtain a large supply of other anti-malarial drugs and among the reports on these is one by Briercliffe[2] on the use of "atebrin" musonate. It is a comparative test of "atebrin" musonate and quinine made in the hospitals during April, 1935. A group of 681 patients was given two intramuscular injections of 0.375 gram of "atebrin" musonate with an interval of 24 hours between them. A second group of 424 patients was treated with two intramuscular injections of 15 grains of quinine followed by quinine given by the mouth. About a quarter of the "atebrin" group and a third of the quinine group complained of pain at the site of the injections; there were two abscesses in the "atebrin" group but none in the quinine group. There were more deaths in the "atebrin" group and 4 of the 17 deaths were attributed directly to "atebrin."

Between May 20 and May 31, 1935, 1387 persons in malarious villages were treated with "atebrin" injections; 61 suffered from toxic symptoms such as vomiting, collapse and abdominal pains. Mental symptoms, which in most cases lasted for about a week, occurred in 8 patients and one of them died from exhaustion. Pain at the site of injection was complained of by most of the patients, and in 9 abscesses resulted. The data available did not allow definite conclusions to be drawn

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with respect to the relapse-rate but the belief exists that a full course of "atebrin" by mouth (1.5 gram) has a greater effect in preventing the return of fever than a week's careful treatment with quinine, and that either of these courses of treatment is better in this respect than two injections of "atebrin" musonate. The majority of patients suffering from malaria do not require treatment by injection with either drug. When, however, treatment by injection is indicated, quinine is to be preferred to "atebrin."

The temporary mental derangement which may result from the administration of "atebrin" proved a serious objection to its use; in one district, where several hundred people had been treated with oral "atebrin," at least 15 cases occurred and the use of "atebrin" was abandoned.

Schulemann[3] who has been closely identified with the development and use of "atebrin" and plasmochin has made an authoritative statement that these two drugs when given together are more apt to cause abdominal pain than when they are given one after the other.

Soesilo and Gilbert[4] tested prophylactic drug treatment in a hyperendemic malarial region using 175 children, 40 of these were under and 135 over 3 years of age, the parasite index at the start was 65.7 per cent. All received the official malaria tablets D. V. G. (each had 240 mg. quinine sulphate and 2 mg. plasmochin) for one week in therapeutic doses after which the prophylactic trial began of the different test drugs for a period of 15 weeks; then came the end of the treatment, but the blood surveys for the parasite index were continued for 28 weeks in all. There were four groups with 43, 44, 43 and 45 children in each: (1) The use of daily therapeutic doses of D.V.G. malaria tablets for 1 week lowered the parasite index of 65.7 per cent to 4.5 per cent. (2) The children of "atebrin" Group 1 remained wholly parasite free (dosage on 3 days in the week) ½ to 1 tablet (50 mg.). (3) Group 2, ½ to 2 tablets (100 mg.) quinine hydrochlorate, and Group 3, ½ to 2 malaria tablets D.V.G. same period and frequency as Group 1. These were never completely free of parasites, but had a much lower parasite index over the whole 15 weeks than before treatment was begun. (4) Group 4 was given anti-beriberi-vitamin tablets as a control group of no antimalaria treatment. This series had a consistently higher index than Groups 2 and 3, but this was, nevertheless, lower than before the trial began. The index in a neighboring town was 65.2 per cent at the close of these experiments. The stoppage of the experiment was followed by a rise of the parasite index in all the test groups. The expected continued action of "atebrin" for two weeks was not clearly established.

Bonne and Stoker[5] made experiments on prisoners in Java. There were four groups each with 75 prisoners:

Group 1 received one tablet of "atebrin" (0.1 gram) every evening.

Group 2 received one tablet of quinoplasmine (0.01 gram plasmoquine and 0.3 gram of quinine).

Group 3 received two tablets of quinine (0.4 gram).

Group 4 control. No drug given.

The drugs were given for three months, and this was followed by an observation period of two months. The results were as follows: no clinical case developed in the *"atebrin" group* in three months. Parasites were found only twice at the weekly thick-film blood examination. Three weeks after the treatment had been stopped, the parasite index was no better than in the control group. There were no toxic symptoms, but at the close of treatment every patient was stained yellow. This raises the question of the cumulation of "atebrin" and the danger of hypersensitivity to the sun following the deposition of acridine in the skin. The *quinoplasmine group* had two clinical cases with 44 hospital days. The *quinine group* had four cases with 50 hospital days, while the *control group* had 21 cases with 243 hospital days.

Peter,[6] in a discussion of the clinical testing of malarial remedies, says that "atebrin" differs fundamentally from quinine and plasmochin in its longer retention in the body. It is a dyestuff and its action on the parasites is in all probability a direct one. It has a marked affinity for the parasites to which it becomes firmly bound and on this account the amount required should be regulated by the number of parasites present rather than by the weight of the patient. Foodstuffs with abundant cellulose absorb "atebin" and thus diminish its effect.

Carman[7] advises that subtertian infections should be treated immediately. It is vain to hope for the development of immunity against it in European cases. "Atebrin" is as effective as quinine and costs no more. His observations were made in Kenya. He does not believe that Europeans ever develop a useful immunity to subtertian malaria. He finds that quinine and "atebrin" are equally efficient in eliminating parasites, but prefers "atebrin" for hospital use since it requires a shorter course and releases beds earlier so that 14 per cent more patients can utilize the space.

Hicks[8] calls attention to the fact that "atebrin" musonate is the methyl sulphonic acid salt of "atebrin." Ordinary "atebrin" is the dihydrochloride. "Atebrin" musonate is sold as "Atebrin for Injection" and must be distinguished from "Atebrin Tablets for Injection," which consist of the dihydrochloride.

Winckel,[9] discussing malarial therapy in the Psychiatric Clinic at Amsterdam, says that he uses an injection of a small dose (150 mg.) of neoarsphenamine to "damp down the symptoms when they tend to get out of hand." They have abandoned the use of provocatives of malarial infections such as epinephrine.

Barbosa[10] reports that in La Bozagona, Spain, malaria has been treated by drugs for the five-year period 1930-1934. The general conclusions were that the intensive treatment of patients suffering from tertian infections did not lessen the incidence by this type in the period under consideration. During that period all subtertian infections were given combined quinine and plasmochin and the numbers attacked increased steadily year by year. Such an increase was not seen when quinine alone was used. Consequently, he finds no advantage in giving the combined drugs.

Franchi and Sautel[11] discuss preventive quinine during military maneuvers in a malarious district in the south of Corsica during 1934. They draw particular attention to the wastefulness of this method. If a French soldier contracts malaria he becomes automatically entitled to a pension, even though preventive quinine may have made the clinical symptoms negligible. Large doses must be given if they are to prevent symptoms; the dose used by the French in Macedonia was 10 grains daily. The advantage of preventive quinine is that the soldier can be kept fit for duty over a special period. When the quinine is stopped the patient experiences a sudden attack of malaria without any stage of invasion; the author calls it "decapitated malaria."

Hill and Olavarria[12] report a three years trial with a short treatment of 1 gram (15.5 grains) of quinine sulphate daily for 4 days in the dispensary at Campo Lugar, a town of 1,200 people in Spain. They think this satisfactory for tertian, but not quite long enough for subtertian infections.

Dawson, Gingrich and Hollar[13] offer the advice that intravenous injections of "atebrin" should be given only in emergency. The injection should be given slowly, and the dose should not exceed 0.1 gram. The toxicity of "atebrin" intravenously is 20 to 40 times greater than that of "atebrin" by mouth.

Vardy[14] gave "atebrin" musonate injections to 50 serious cases. In 32 the patients were cured in 48 hours, in two the drug had no effect and quinine was given while in two others the drug may have had a toxic effect. He states that intramuscular injections are to be preferred over the intravenous method.

Barbosa[15] reports on the use of quinine and "atebrin" in preventing recrudescences after recurrence of benign tertian malaria. He accepts James'[16] definition of "recrudescence" as a return of fever and parasites within 8 weeks of recovery from the primary attack; a "relapse" as a return between 8 and 24 weeks, and a "recurrence" as a return at some time later than 24 weeks. Barbosa treated 49 cases of benign tertian with quinine and a similar number with "atebrin." In each group there were 12 adults and 37 children. The dosage of "atebrin" was scaled from 0.1 to 0.3 gm. daily according to the age of the patient. Quinine was also graded, the adult dose being 1 gm. daily. Each patient was kept under observation for a period of two months following recovery from the recurrence. Seven (58.3 per cent) of the 12 adults on quinine showed a recrudescence, while the "atebrin" series had five, or 41.6 per cent. Among the children on quinine the rate was 48.6 per cent and for "atebrin" 37.8 per cent. In other words, he believes "atebrin" is more effectual than quinine in preventing recrudescence in benign tertian malaria. "Atebrin" delays the appearance of the recrudescence, the interval being fully twice as long as when treated with quinine.

Kirilow-Drenowsky,[17] in Bulgaria, presents some observations on a 6-day treatment with "atebrin," "atebrin" and plasmochin simplex, plasmochin Co., quinoplasmochin and quinine. He reports the method of choice to be the "atebrin" plasmochin combination, which gave only 3.7 per cent of relapses. He placed next the "atebrin" treatment alone. Next in order were quinine (25 per cent relapses), quinoplasmochin 33 per cent, and plasmochin Co. 42 per cent relapses.

Hicks and Chand[18] treated 210 prisoners in India suffering from benign tertian and 158 with malignant tertian. The drugs used were (a) quinine, (b) totaquina Type 1 (32 per cent quinine and 11 per cent cinchonine), (c) totaquina Type 2 (19 per cent quinine and 20 per cent cinchonine). The doses, 0.6 gram daily per 70 kg. of body weight in tertian and 1.2 gram daily in subtertian. The drugs were given in tablet form. The patients had spent their lives in an endemic area of India. The results were practically the same, a mean duration of parasites of 1.5 days, and of fever, 2 days. They suggest that a dose of 15 grains once daily for 3 or 4 days would be suitable for the routine treatment of rural populations in the Punjab. This would remove the clinical symptoms, which is all the people demand.

Pärvulescu and Boerin[19] made comparative tests with two types of totaquina and with quinine sulphate. The patients were 213 young soldiers. Benign tertian cases were given 9 grains daily for 5 days, subtertian cases received 18 grains daily. Totaquina was less efficient at these doses, but when double the quantity was used they were as effective as quinine.

Marañon, Perez and Russell[20] report on the use of totaquina in the Philippines. Cinchona with an excellent alkaloidal content can be cultivated in the Philippines, and a satisfactory totaquina can be prepared locally and sold at a seventh of the present price of quinine. Refined quinine is an expensive luxury that can be used as treatment only by the rich patient. Totaquina is an excellent drug for the poor. The production and use of totaquina would not interfere with the usual sale of other antimalarial drugs.

Vigoni[21] reports on the use of quinine-Weil in the Belgian Congo. The formula for this drug is C_{20} H_{24} O_2 N_2 C_{16} H_{11} O_2N. It is free from the bitter taste of quinine. He gave from 10 to 50 centigrams per day, according to body weight. He concludes that quinine-Weil (60 per cent quinine base) appears to be very similar to quinine dihydrochloride in its action. Its main advantage is absence of the bitter taste of quinine and milder toxic reaction.

Massias, Bourgin and Nguyen-Van-Tan[22] report experience with quinacrine and rhodoquine. They report excellent results without toxic symptoms. The reader is referred to the article for details.

Chopra, Sen and Ganguli[23] report the use of "tebetren" in India. It resembles quinine, but costs more. It is a combination of acridine and quinine derivatives with a derivative of cholic acid. They treated 22 patients with 3 tablets three times a day for five days and then continued observations for a fortnight. They found that it resembles quinine and "atebrin" in action, but costs more and offers no particular advantage over them.

Clark and Komp[24] present a sixth year's report on Chagres River malaria, where field experiments have been continued with "atebrin" plasmoquine simplex in one group, quinine-plasmochin simplex in a second group with a third group for control, but the control has access to quinine for voluntary use.

The effectiveness of the two drugs are considered the same, but the people prefer to take "atebrin." We have had no instances of mental derangement in the use of "atebrin" for the three years it has been given a trial. The present year includes 117 adults and 127 children, and many of these have had several courses of treatment for relapse, and in these cases we give a seven day course instead of a five-day course. Plasmochin simplex is given after the "atebrin" course has been completed. We can keep the parasite index at almost a constant level of 9 per cent, but there is little hope of lowering it without additional antimalarial measures being instituted. Our last year's experience shows that we cannot prevent an epidemic, but the effects of the epidemic can be held in control in so far as serious clinical cases are concerned.

The bulk of our malaria this year, as in the past few years, is confined to the same individuals or families, yet some of these individuals will average a course of treatment every other month.

It would be impossible in a hyper-endemic area such as the Chagres to apply James' idea of recrudescence, relapse and recurrence, but we feel that the bulk of our cases represent chronic carriers of the disease rather than new infections. A study of babies during the first 12 months of life supports this belief. We were able to get 12 consecutive monthly surveys on 259 people this year. One hundred four (40.1 per cent) were negative throughout the year. One hundred fifty-five (59.8 per cent) were positive at least once during the year and 58 (37.4 per cent) of these positives remained negative after treatment for the remainder of the year, but 97 (62.6 per cent) developed either reinfection or relapse from one to eight times during the year. We think they were chiefly the reappearance of the old infection. The peak of the disease is among persons from 5 to 20 years

of age. It is interesting to note that we had in our group this year 68 persons over 60 years of age and 18 of these (26.5 per cent) revealed parasites in their films one or more times and two of them were clinical cases. All of these people have spent their lives in this same area. They, of course, have a very respectable degree of tolerance, but statements regarding immunity to malaria should be given careful thought. We do not fear that drug control over a period of years will deprive people of an opportunity to carry light or chronic infections, although in the past we have shared that opinion. Eradication of a malarial infection and control of one are two very different things. Our antimalarial drugs are wonderful possessions, but they cannot, alone, eradicate all infections. For the past ten years those who practice transfusions[25] have been producing facts and cases of malaria from symptomless carriers of malaria that have extended over periods of years. Bastianelli[26] thinks that the current treatment of malaria does not interfere with the immunizing processes that lead to final cure. He emphasizes the time factor necessary for the acquisition of immunity and discusses the view that, in a hyperendemic district, so-called radical treatment may reduce or prevent resistance to infection.

REFERENCES

1. Watson, Malcolm: Some Pages from the History of the Prevention of Malaria. Glasgow Med. Jour., **5**: Nos. 2, 3, 4, and pp. 49 70, 130-153, 202-220, 1935.
2. Briercliffe, R.: The Ceylon Malaria Epidemic, 1934-35. Report by the Director of Medical and Sanitary Services. Sessional Paper XXII, Sept., 1935. Colombo: Govt. Record Office
3. Schulemann, Professor: Recorded by W. F. in Abstracting Malaria and Its Treatment (Brit. Med. Jour., 1935, Sept. 21, p. 552) for Trop. Dis. Bull. **33**:230.
4. Soesilo, R.: and Gilbert, A. Ph. W.: Prophylactic Drug Treatment in a Hyperendemic Malarial Region. Geneesk. Tijdschr. v. Nederl.-Indie, **75**:1141-1145 (July) 1935.
5. Bonne, W. M.; and Stoker, W. J.: Malaria Prophylaxis with Atebrin, Quinoplasmine and Quinine. Geneesk. Tijdschr. v. Nederl.-Indie, **75**:958-969 (June 11) 1935.
6. Peter, F. M.: The Clinical Testing of Malarial Remedies. Trans. Roy. Soc. Trop. Med. & Hyg., **29**:41-50 (June 29) 1935.
7. Cerman, J. A.: Atebrin, Plasmoquine and Quinine in the Treatment of Malaria. Trans. Roy. Soc. Trop. Med. & Hyg., **29**:191-202 (July 31) 1935.
8. Hicks, E. P.: Atebrin Musonate: A Note on the Rate of Absorption and on the Local Effects of Intramuscular Injection. Records of the Malarial Survey of India, **5**:203-206 (June) 1935.
9. Winckel, Ch. W. F.: Malaria Therapy. Psychiatrische en Neurologische Bladen, No. 3, 1935.
10. Barbosa, Amando: Fighting Malaria by Drugs in the Absence of Antimosquito Measures. Riv. di Malariologia, Sez. 1, **14**:352-368, 1935.
11. Franchi, F.; and Sautel, J.: Preventive Quinine During Military Maneuvers in a Malarious Country. Bull. Soc. Path. Exot, **28**:456-460 (June 12) 1935.
12. Hill, Rolla B.; and Olavarria, Jose: The Treatment of Malaria with Small Amounts of Quinine. J.A.M.A., **104**:2329-2332 (June 29) 1935.
13. Dawson, W. T.; Gingrich, Wendell; and Hollar, E. D.: Intravenous Toxicity of Atebrine (atebrin). Amer. Jour. Trop. Med., **15**:515-520 (Sept.) 1935.
14. Vardy, E. C.: Notes on a Clinical Investigation of the Treatment of Malaria by Atebrin Musonate Injections. Malayan Med. Jour., **10**:67-77 (Sept.) 1935.
15. Barbosa, Amando: Quinine and Atebrin in Preventing Recrudescences After Recurrence of Benign Tertian Malaria. Medicina Pafes Calidos, Madrid, **8**:139-144 (March) 1935.
16. James, S. P.: Some General Results of a Study of Induced Malaria in England, Abstracted by H. H. S. in Trop. Dis. Bull., **32**:744.
17. Kirilow-Wrenowsky, A.: Comparative Therapeutic Observations on a Six-Day Treatment with Atebrin, Atebrin + Plasmoquine Simplex, Plasmoquine Co., Quinoplasmoquine and Quinine. Arch. f. Schiffs-u. Trop.-Hyg., **39**:243-252 (June) 1935.
18. Hicks, E. P.; and Chand, Diwan: The Relative Clinical Efficacy of Totaquina and Quinine. Records of the Malaria Survey of India, **5**:39-50 (March) 1935.
19. Parvulescu, Constantinesco N.; and Boerin, V.: Totaquina in Malaria: Comparative Worth. Arch. Roumaines Path. Experim. et Microbiol., Paris, **7**:523-528 (Dec.) 1934.
20. Maranon, Joaquin; Perez, Mando; and Russell, P. F.: Philippine Totaquina. Philippine Jour. Sci., **56**:229-255 (Mar.) 1935.
21. Vigoni, M.: On Quinine-Weil in the Treatment of Malaria. Arch. f. Schiffs-u. Trop.-Hyg., **39**:416-422 (Oct.) 1935.
22. Massias, Charles; Bourgin, T.; and Nguyen-Van-Tan: The Treatment of Malaria by an Acridine Derivative (Quinacrine) and a Quinoline Derivative (Rhodoquine). Bull. Soc. Med-Chirurg., Indochine, **13**:79-82 (Feb.-March) 1935.
23. Chopra, R. N.: Sen, B.; and Ganguli, S. K.: Tebetren in Indian Strains of Malaria. Indian Med. Gaz., **70**:362-366 (July) 1935.
24. Clark, H. C., and Komp, W. H. W.: A Sixth Year's Report on Malaria in Panama (Chagres Valley) with Reference to Drug Control. Read before National Malaria Committee, November, 1936.
25. A Malarial Relapse After Nine Years. Transmission to the Donor During Transfusion. The Danger of Transfusion in the Colonies. Rev. Med. et Hyg. Trop., **23**:32-37 (Jan.-Feb.) 1931.
26. Bastianelli, G.: Respective Roles of Immunity and Treatment in the Care of Malaria. League of Nations Report, Geneva, C. H. Malaria 238, May 12, 1936.

RESEARCH UPON THE MALARIA PLASMODIA, 1935-1936*

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HUMAN MALARIA

Boyd[1] studied the schizogonous cycle of *Plasmodium vivax* in an attempt to differentiate schizonts which gave rise to asexual parasites from those which gave rise to gametocytes. He found five series of cells. Series A were interpreted as the source of trophozoites, but in certain of these schizonts a single merozoite different in form from the others was interpreted as giving rise to a schizont of either Series B or D which were interpreted as pregametocytes. The schizogony of these parasites, he believed, gave rise to Series C or E, representing male and female gametocytes, respectively.

In support of these observations, Boyd, Stratman-Thomas and Muench[2] studied the gametocyte formation of *P. vivax* during the primary attack. They found gametocytes in the blood only after the fourteenth day following inoculation, after which there was a gradual increase in density up to about the fifth week following inoculation, and then a decline. Upward trends in number appeared with great regularity at approximately five-day intervals, corresponding with the maturation of the gametocyte series found in the morphological study of the parasites.

Corradetti[3] attempted to confirm Boyd's observations, but found only two types of schizogonous rosettes which showed a different arrangement of pigment and merozoites with different nuclear and cytoplasmic characters of the merozoites. He also found two types of parasites of the earliest stage having characters respectively similar to those of the merozoites in the rosettes. Certain rosettes contained a merozoite with different

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staining properties from the others, but he did not establish these as the source of the second of his two types of rosettes. He stated that a differentiation of schizogony stages on a morphological basis is not yet justified.

Thomson and Robertson[4] reviewed previous observations concerning the development and structure of the gametocytes of *P. falciparum*. From specimens received from Southern Asia and from Africa they described the development of these forms and confirmed the observations of others that a capsule or periplast as an ectoplasmic structure of the organism appears during its development, being most prominent in half-grown crescents and later becoming more delicate so that it is difficult to demonstrate by ordinary staining methods.

Casini[5] studied the development of gametes from gametocytes in *P. vivax, P. falciparum* and *Halteridium danilewski*. The study was made with fresh blood, blood preserved in a humid chamber, and blood extracted from the stomach of Anopheles mosquitoes. He believes that gametocytes do not attain maturity by the expulsion of polar bodies as has been generally believed. His observations indicated that in both male and female gametocytes the chromatin not used in fecundation becomes separated from that which gives rise to the male and female elements. In the female gametocyte it is expelled like a foreign body, while in the male gametocyte it remains as a residual body after the formation of the flagellate gametes. He believes that all other formations which he observed in his preparations and which have heretofore been considered polar bodies consist of residues of erythrocytes or deformation of the body of the parasite.

Ivanic[6 7 8] observed blood smears containing *P. vivax* for evidences of mitotic nuclear division. His first observation showed evidence that the first stage of nuclear division was promitotic in character. His second observation showed a similar form of division in one of two daughter chromatin masses, and his third observation showed a true mitosis in the mother nucleus. From these observations he concludes that the malaria parasite originated from a more elementary rhizopod in which mitosis was the rule, and that infection may have originally been acquired by mouth rather than by the bite of mosquitoes.

Knowles and Basu[9] studied the nuclear division of sporozoites of *P. vivax* and *P. falciparum* obtained from *Anopheles stephensi*. They found various stages of division of the original chromatin into two masses and the division of one of these forming three separate nuclei. They considered that these findings corroborated observations of several previous observers who placed no significance on the finding, and also the observations of Missiroli working with *P. relictum*, who described division of the sporozoite into rounded bodies which applied themselves to the surface of red blood corpuscles and presumably developed into ring-trophozoite forms. They stated that these findings indicate that the sporozoite may not be the end-phase of the sporogony cycle.

Jacobsthal[10] stained malaria blood films with Loeffler's methylene blue in order to study the red cells infected with *P. vivax* and *P. falciparum*. He corroborated the findings of Eaton which I reviewed two years ago and those of Brug which I reviewed last year, showing that 90 to 98 per cent of the infected erythrocytes were recticulocytes. He noted that the reticular structure of infected cells was altered. He considers that the parasitization of these young erythrocytes is of clinical importance because their destruction hastens the development of anemia.

Sarkissiane[11] studied the development of Schueffner's granules in erythrocytes parasitized by *P. vivax*, using Nile-blue sulphate as a vital stain, and subsequent fixation and staining with azure-eosin. He found that granulation started in the ring stage, reached the maximum in half grown schizonts, and disappeared little by little as the parasite matured. Meanwhile the superficial layer of the erythrocytes became damaged and finally lost, the cells became swollen and lost their hemoglobin and the remaining stroma became raspberry colored with azure-eosin. He concluded that the superficial portions of the erythrocytes were used in the nourishment of the parasites. The pigment of the parasites was partially stained red by Nile-blue sulphate which the author interpreted as evidence of the presence of liprochrome in the pigment.

Barber[12] observed degeneration of sporozoites of *P. vivax* and *P. falciparum* in the salivary glands of Anopheles mosquitoes in Greek Macedonia and in Cyprus. Most of the observations were carried out with *Anopheles elutus* and *A. superpictus*, both laboratory infected and naturally infected. Degeneration of the sporozoites was found to be definitely more common in *superpictus* than in *elutus*. Even comparatively young sporozoites at summer temperatures sometimes showed degeneration. The degree of degeneration often varied in the two salivary glands of the same mosquito or in two lobes of the same gland. No factors were discovered which seemed to be definitely correlated with the presence of degeneration. Epidemiological studies indicated that degeneration of sporozoites may help to explain the relative inability of *A. superpictus* to transmit malaria, especially in some parts of Macedonia.

Boyd, Kitchen and Muench[13] investigated the geographical distribution in nature of the McCoy strain of *P. vivax* which they have used in their biological studies of malaria in northern Florida. Their investigation was based upon evidence of immunity or susceptibility to this strain in paretics to which the parasite was applied therapeutically. Patients from the western part of Florida and the contiguous part of Alabama presented the shortest average duration of attack and those from western Florida showed the lowest percentage of takes on first inoculation. These facts, together with the detection of a person from this region entirely refractory to this strain and the fact that the strain was originally isolated from western Florida, led to the conclusion that this area is the indigenous habitat of the strain.

MONKEY MALARIA

Pandit[14] compared the results of fixing blood films containing monkey malaria parasites by air drying with wet fixation in osmic acid vapor. Both types of fixation were followed by secondary fixation in methyl alcohol, after which Giemsa stain was applied. He stated that osmic vapor fixation produced better demonstration of the amoeboidicity of the parasites. The chromatin mass appeared to be more mobile and more spread-out. Two vacuoles could be made out in many of the forms, one surrounded by pink staining chromatin, the other by blue staining cytoplasm. Furthermore, certain very peculiar forms of *P. cynomolgi* were seen, in

which the nuclear vacuole, instead of being completely surrounded by chromatin, appeared to open out into the erythrocyte through a cleft in the chromatin. The erythrocyte also appeared smaller and more purplish after osmic vapor fixation. Pandit believes that this method of fixation kills the parasite more rapidly and, therefore, fixes it in a more natural condition.

BIRD MALARIA

Huff and Bloom[15] found that *P. elongatum* is capable of living in all of the blood and hemopoietic cells of the canary in contrast to other known species of avian plasmodia. Parasites were studied in smears made from the cut surfaces of liver, spleen and bone marrow. The smears were fixed in Zenker-formol and were stained with Delafield's hematoxylin followed by eosin-azure II. Other methods of fixation and staining were also used for the study of various types of cells. All of the asexual stages of the parasite were found in all of the blood and blood-forming cells. The vast majority occurred in cells of the erythrocyte series. The authors considered it probable that the parasites in phagocytic cells were destined for destruction since both living and degenerated parasites were found in these cells, whereas only normal appearing parasites were found in cells of the erythrocyte series. Normal parasites were also found occasionally in the nonphagocytic plasma cells, indicating that this cell offered a suitable medium for the progressive development of the parasite. Gametocytes were found only in normoblasts and erythrocytes. Only the parasites in cells with obvious hemoglobin produced pigment. Although the morphology of some of the developmental stages of the parasites were found to be different from the same stages of other species, the authors believe that these differences are insufficient to exclude it from the genus *Plasmodium*. The merozoites were found to be elongate in shape, suggesting a close relationship between the Suborders Haemosporidiidea and Eimeriidea. This study also demonstrated that *P. elongatum* had quotidian periodicity with segmentation occurring in the morning hours in contrast to *P. cathemerium* in which segmentation is restricted to the evening hours.

Brumpt[16] described *Plasmodium gallinaceum*, a new species from the domestic fowl. This plasmodium produces from 8 to 32 merozoites. The nucleus of the erythrocyte is displaced both by schizonts and gametocytes. No attempts were made to transmit this plasmodium to other species of birds.

Wolfson[17] described *Plasmodium oti*, a new species from the eastern screech owl. The characteristics of this species are as follows: gametocytes elongate, not displacing the nucleus of the erythrocyte but tending to bend around both poles of the nucleus; schizonts elongate, not displacing the nucleus, located usually parallel to the long axis of the cell, number of merozoites usually eight, pigment granular to rod-like. The parasite is the first plasmodium from an owl successfully transmitted to canaries. The author also states that the common species of plasmodium occurring in owls and having round gametocytes is considered to be *P. subpraecox*.

Manwell[18] succeeded in transmitting *P. polare* from the cliff swallow to the canary. Eight birds were infected and the parasite was carried through five transfers. All infections were extremely light, but no case of spontaneous elimination of the infection was observed. There was no evidence that the parasite was at all pathogenic to the canary.

Wolfson[19] studied the synchronicity, periodicity and length of the asexual cycle of *P. rouxi* in the canary. She defined synchronicity as the degree of concurrence of any designated stage at any particular time, and periodicity as the degree of similarity in the length of the different asexual generations. She concluded that the degree of synchronicity in this plasmodium is low since any growth stage could be found in the blood at practically any time of day. Periodicity, on the other hand, is marked, and the length of the asexual cycle appears to be 24 hours, since there was no evidence of sporulation at longer intervals.

Wolfson[20] also studied the periodic phenomena of *P. circumflexum* in canaries. She found the synchronicity relatively high since at certain times there was complete lack of some stages and the large predominance of others. She found the length of the asexual cycle to be about 48 hours, the largest amount of sporulation occurring during the late afternoon hours.

Schwetz[21] studied plasmodia from an owl and a weaver bird in the Belgian Congo. The owl was found to harbor a *Haemoproteus* and also two plasmodia, which he identified as *P. praecox* (*relictum*) and *P. fallax*, the latter having been originally described by him in 1930. The plasmodia of the weaver bird were identified as *P. praecox* (*relictum*) and *P. elongatum*.

Demidowa[22] injected decreasing numbers of *P. praecox* into birds, using micromanipulation with a dark field condenser. Successful inoculations producing infection were obtained even by the injection of a single infected erythrocyte. By reducing the dose of the infected material the normal incubation period of 5 to 7 days became prolonged to 10 to 52 days.

REFERENCES

HUMAN MALARIA

1. Boyd, M. F.: Amer. Jour. Trop. Med., **15**:605-629, 1935.
2. Boyd, M. F.; Stratman-Thomas, W. K.; and Muench, H.: Amer. Jour. Trop. Med., **16**:133-138, 1936.
3. Corradetti, A.: Riv. di Malariol., **15**:14-22, 1936.
4. Thomson, J. G.; and Robertson, A.: Trans. Roy. Soc. Trop. Med. & Hyg., **29**:31-40, 1935.
5. Casini, G.: Riv. di Malariol., **15**:88-98, 1936.
6. Ivanic, S.: Zent. f. Bakt. Parasit. u. Infekt. I. Orig., **133**: 274-282, 1935.
7. Ivanic, S.: Zent. f. Bakt. Parasit. u. Infekt. I Orig., **136**: 109-111, 1936.
8. Ivanic, S.: Zent. f. Bakt. Parasit. u. Infekt. I. Orig., **136**: 418-420, 1936.
9. Knowles, R.; and Basu, B. C.: Ind. Jour. Med. Res., **22**: 443-447, 1935.
10. Jacobsthal, E.: Klin. Woch., **15**:942, 1936.
11. Sarkissiane, A. B.: Folia Haematol., **54**:145-149, 1935.
12. Barber, M. A.: Amer. Jour. Hyg., **24**:45-56, 1936.
13. Boyd, M. F.; Kitchen, S. F.; and Muench, H.: Amer. Jour. Trop. Med., **16**:583-587, 1936.

MONKEY MALARIA

14. Pandit, S. R.: Rec. Malar. Surv. India, **5**:389-392, 1935.

BIRD MALARIA

15. Huff, C. G.; and Bloom, W.: Jour. Inct. Dis., **57**:315-336, 1935.
16. Brumpt, E.: Compt. Rend. Acad. Sci., **200**:783-785, 1935.
17. Wolfson, F.: Amer. Jour. Hyg., **24**:95-101, 1936.
18. Manwell, R. D.: Jour. Parasitol., **22**:412-413, 1936.
19. Wolfson, F.: Amer. Jour. Hyg., **23**:341-348, 1936.
20. Wolfson, F.: Amer. Jour. Hyg., **24**:207-216, 1936.
21. Schwetz, J.: Riv. Malariol., **14**:215-247, 1935.
22. Demidowa, L. W.: Giorn. d. Batter. e Immunol., **13**:872-877, 1934. Abstr. Trop. Dis. Bull., **33**:237-238, 1936.

SOME RECENT ADVANCES IN THE EPIDEMIOLOGY OF MALARIA*

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The Sub-Committee on Epidemiology as in former years has adhered to the practice of confining its report to studies outside the continental United States. A report of this type obviously cannot cover more than a small part of the literature on this subject.

Last year your attention was called to an article by Sinton.[1] Since that time, he[2 3] has completed this most interesting and convincing article, "What Malaria Costs India Nationally, Socially, and Economically." In addition to other costs, he points out that malaria causes a tremendous loss to the government in the decrease in revenue and the increase in cost of operation.

James[4] and Gill[5] give excellent reports of the epidemic of malaria in Ceylon. Ceylon is about one-half the size of England with about three and one-half million population. The humid fertile southwestern section contains 60 per cent of the population. *A. culicifacies* is the only active carrier among many species of anopheles, and benign tertian is the type of malaria commonly present. There are only a few cases of malignant tertain. The northeastern section is dry and normally has a splenic index of 70 per cent as compared with 5 per cent in the humid area. It had been previously noticed that in dry years when the monsoon failed, malaria increased in the humid area. In 1934, the monsoon was absent. Two weeks later, in early October, the epidemic started and in November had increased to ten times the normal number of cases. The epidemic reached its height about the middle of December, and the first wave declined after that and ended in March, but was quickly followed by a second wave. The case mortality was low, being from 1.5 to 3.0 per cent. Of the mosquitoes caught in houses, 14 per cent were infected. James[4] states that the second wave was due to recurrences and not to fresh infections. Benign tertian was the predominating type. He states that the production of late recurrences is characteristic of benign tertian. Gill,[5] on the other hand, believes that, as the second wave was associated with a rise in the infection rate of anopheles, it was a true epidemic wave, being due in part to new infections as opposed to relapses. This epidemic was due to: (1) drought which changed large lakes and rivers into small shallow clear pools and caused the failure of crops, resulting in poverty and malnutrition; (2) a great multiplication of mosquitoes; and (3) an available immune population after exceptionally healthy years; and Gill[5] adds the presence of "factor X" which occurs periodically every five years and is associated with the malaria parasite. The epidemic cost eighty thousand lives in seven months.

Covell and Baily[6] report observations made on a regional epidemic of malaria in Northern Sind. They point out the similarity between this epidemic and that of Ceylon. It was marked by a great increase in the number and infection rate of mosquitoes. It was among the population with a low communal immunity. The economic conditions were similar. Ceylon had no crops due to drought, while Sind's poverty was due to an epidemic of cholera and the occurrence of floods with the resultant loss of property. Malignant tertian predominated in Sind; whereas, benign tertian was the predominant type in Ceylon. They note that the conditions favorable to propagation were longevity and activity of anopheles and low immunity. The latter is necessary for high mosquto infection, as the gametocyte count is high in these and not in those with a higher degree of immunity.

These same workers[7] report that Sind was highly malarious before the Lloyd barrage scheme commenced operation and was subject to periodic epidemics of malaria, especially in the northern area. Since the operation of this system of canals, malaria has become considerably worse due to a rise in the subsoil water levels with waterlogging, to seepage from some of the new canals, and to the cutting off and impounding of old canals, all of which resulted in prolific breeding of mosquitoes. They further call attention to the fact that there has been a great increase in the amount of rice fields because of the availability of water.

Timbers,[8] reporting a study covering two malaria seasons in Western Bengal, notes that the region is hyperendemic with wide variations in intensity for villages and years. *P. vivax* is the most prevalent, with *P. falciparum* second during the fall and *P. malariae* second in the spring. Kala-azar is present and complicates the spleen picture. *A. philippinensis* is the chief vector in summer and fall. *A. annularis* (he notes that this is synonymous with *fuliginosus*) and *A. pallidus* are the only other species of importance. *A. pallidus* is the only one infected during the cold weather preceding the spring epidemic. *A. philippinensis* was reduced and not infected, while *A. annularis* was plentiful but not infected. Gametocytes were present and plentiful throughout the year, but were most frequent in December. The sporozoite rate was highest from July to October. Each of these anopheles had its greatest intensity one to three months later; that is, the period of optimum infectivity precedes the season of its greatest density. The three carriers breed only in still, clear, shady water with lots of aquatic vegetation. Rice fields were found not to be important breeding places. *A. culicifacies, A. minimus,* and *A. aconitus,* which are efficient carriers elsewhere, are not carriers in Western Bengal. They are scarce and not infected.

Christophers[9] calls attention to the fact that a malaria survey is of the highest importance and points out that it is impossible, until actual facts are known, to determine which, if any, measures of prevention are to be adopted. He points out that in Bombay, *A. superpictus* was the most common anopheles and would have been controlled; but the malaria survey showed them to be harmless, and *A. stephensi,* breeding in wells and cisterns, was found to be the vector.

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Hackett[10] points out, in a study of the biological factors in malaria control, that there are two epidemiological types of malaria in Europe. One is due to the presence of races or species of anophelines which habitually feed on man. These are dangerous in strict proportion to their numbers. The other type depends on conditions permitting contact between anopheles and man which is usually suppressed through natural biological influences. In old and progressive agricultural communities, there is no relation between the numbers of these anopheles and the amount of malaria. He states that animal barriers are never useless, but that in the case of dangerous anophelines, animals do not deviate them so much as they serve to dilute the blood supply by adding a large number of unsusceptible hosts.

Garnham[11] states that malaria in an African in a hyperendemic area causes little sickness. Preventive drug treatment, therefore, serves no purpose. A study of malaria in the native reserve of Kenya shows that 85 per cent of the children are infected by the fifth month and 100 per cent by the sixth. These children are well nourished and healthy. Malignant tertian and quartan are ubiquitous. Benign tertian is rare but has a focal distribution and is the most dangerous parasite. *P. falciparum* and *P. vivax* appear by the end of the second month of life, and *P. malariae* not until the fifth month. By the end of the first year, nearly every child is infected with *P. malariae*, but immunity against this parasite is soon established. Only 1½ per cent of the adults were infected with this type. Fifty per cent of the infants two months old, 80 per cent of the children, 30 per cent of the young adults, and 25 per cent of the older ones had crescents.

Wickramasuriya[12] reports six cases of congenital malaria. In the first three cases, massive infection of the placenta with malignant tertian was noted. The first case had a few malignant tertian parasites in the umbilical cord. The baby's temperature rose on the fifth day. In the second and third cases, there was intrauterine death of the fetus and malignant tertian was found in the fetal brain and spleen. In the fourth case, the mother died of malignant tertian. Pigment only was found in the fetus. In the fifth case, the mother died of malignant tertian and parasites were exhibited in the fetal heart, liver, and spleen. In the sixth case, the mother died with malaria and crescents were found in the blood of the cord, but not in the fetal liver or spleen.

Bourgin[13] reports a case with *P. vivax* gametocytes in the peripheral blood of the mother and in the umbilical cord of the child. Neither the mother nor child had fever. The mother was treated six days after parturition. No parasites were found in the mother or child, but both had a high percentage of mononuclears.

Tanner[14] reports a congenital case of malaria in one of twins. The mother came from India to England. She had a few attacks during pregnancy, the last about ten days before delivery. When the child was 53 days old, *P. vivax* was found and was associated with an enlarged spleen but no fever.

Mackay[15] states that a considerable proportion of children are born with definite evidence of malaria infection contracted *in utero*. He reports that of 44 blood smears taken at birth, 8 showed sub-tertian trophozoites and one benign tertian gametocyte. In this connection, Daleas[16] reports that Henry's reaction is often positive in newly born children of Indo-China, and he believes that congenital malaria is common, though parasites are rarely found in the peripheral blood. He says that parturition and abortion in severe, acute, or chronic malaria without treatment may result in collapse, but that parturition rarely brings on an attack in a case of latent malaria.

The salivary glands of two varieties of *maculipennis* are differentiated by de Buck and Swellengrebel.[17] In *messeae*, the glands are typically rod shaped and frequently contain masses of needle-like crystals in the lateral lobes; while in *atroparvus*, they are club-shaped and devoid of crystals.

These same authors[18] report that *A. maculipennis* is an indifferent vector in Holland in the summer. This is not because they are bad carriers, nor is it due to animal attraction only. It is because of the short life of the summer generation. They found that from September to December the rate of survival was 59 per cent; from January to March, 32 per cent; and from April to August, 15 per cent. The sporozoite rates during these three periods were approximately the same—74, 71 and 70 per cent, respectively. They noted that the heaviest infected mosquitoes live the shortest time and that intestinal infection alone was not apparently responsible for this fact.

Trensz[19] says that if Henry's reaction is due to the presence of antibodies in the blood of the malaria patient as the result of the antigenic presence of hemozoin, then hemozoin should serve as a better antigen than melanin. He found that hemozoin does not act as an antigen in Henry's reaction. According to Prudhomme,[20] the serum of the malaria patient has no specific affinity for melanin. Melanin serves only as an indicator of the stability of serum in distilled water. He states that many other substances can be used in place of melanin with advantage, and he recommends the use of a fine suspension of carmine in distilled water.

Sydenstricker,[21] after trying various vital stains for malaria parasites, found that a saturated solution of brilliant cresyl blue in sterile physiological saline is best. The advantages are that it stains at once and that the parasites remain alive for about two hours on the warm stage.

Pawan[22] states that stained malaria slides may be kept in good condition even in tropical climates. His method of preserving the slides is to dip them in a saturated solution of paraffin wax in xylol. To examine them later, they are cleaned with xylol and examined in the usual way. After examination, the oil is removed with xylol, they are dipped as before, and stored.

Kehar,[23 24] in two reports, makes some very interesting observations on the absorption and excretion of "atebrin." He notes that "atebrin" first appears in the urine in from 15 to 60 minutes, and that 49 to 69 per cent of the administered dose is excreted in from 16 to 28 days. He states that "atebrin" is eliminated from the body over a period of 54 to 69 days. With increase in the amount of urine, there is increased excretion of "atebrin." The absorption and excretion of "atebrin" were found to be slower if taken at meals or two hours after meals than if taken one hour before or two and one-half to three hours after meals.

Helferich[25] and others called attention to the unusual

fact that in a chronic endemic area the parasite index was often found to be much higher than the spleen index. In many instances this was noted as a permanent condition and was found to be equal among all age groups. Helferich believes that this may be due to a low virulence or a strain resistant to medication.

In studying two strains of *P. vivax* used in the treatment of paresis, de Buck[26] found them to differ as regards the average incubation period, the number of parasites present, and the number of merozoites produced per schizont. He found these characteristics to be constant regardless of the method used in inoculation and he lists them as true inherent characteristics of the strains in question.

Cassuto[27] describes two types of relapse. The first he describes as a recrudescence where a number of parasites survive treatment and continue to proliferate. In the second he says parasites are present, but due to immunological equilibrium, they do not proliferate until this equilibrium is upset by an operation or injury or in nature by some intercurrent illness or depressant.

In a study of *A. maculipennis* vars. *labrachiae* and *messeae*, Putoni[28] found that there is no foundation for the idea that anopheles need clear water for breeding. He reports that sewage as a fertilizer would accelerate anopheles breeding and increase the percentage of larvae that grow to adults.

Wilson[29] finds that *A. maculatus* caught in houses represents the number seeking blood. This species is most active in seeking blood during the period when the wet-bulb temperature is high.

Lutrario,[30] in Italy, reports a rhythmical rise and fall in malaria, with small cycles which coincide with the yearly seasons, larger ones with periods of four to five years, and others yet larger whose duration is measured in centuries.

REFERENCES

1. Sinton, J. A.: Rec. Mal. Surv. India, **5**:223-264 (Sept.) 1935.
2. Sinton, J. A.: Rec. Mal. Surv. India, **5**:413-489 (Dec.) 1935.
3. Sinton, J. A.: Rec. Mal. Surv. India, **6**:91-169 (March) 1936.
4. James, S. P.: Bull. Office internat. d'hyg. pub., **27**:1135-1140 (June) 1935.
5. Gill, C. A.: Trans. Roy. Soc. Trop. Med. & Hyg., **29**:427-480 (Feb.) 1936.
6. Covell, G.; and Baily, J. D.: Rec. Mal. Surv. India, **6**:411-437 (Sept.) 1936.
7. Covell, G.; and Baily, J. D.: Rec. Mal. Surv. India, **6**:387-399 (Sept.) 1936.
8. Timbers, H. G.: Rec. Mal. Surv. India, **5**:345-370 (Dec.) 1935.
9. Christophers, S. R.: Riv. di Malariologia (sez. II, No. 3 supp.), **14**:89-95, 1935.
10. Hackett, L. W.: Amer. Jour. Trop. Med., **16**:341-352 (May) 1936.
11. Garnham, P. C. C.: Trans. Roy. Soc. Trop. Med. & Hyg., **29**:167-186 (July) 1935.
12. Wickramasuriya, G. A. W.: Jour. Obst. & Gyn. Brit. Emp., **42**:816-834 (Oct.) 1935.
13. Bourgin, P.: Bull. Soc. med.-chir. de l'Indochine, **13**:137-138 (Feb.-March) 1935.
14. Tanner, N. C.; and Hewlett, R. F. L.: Lancet, pp. 369-370 (Aug. 17) 1935.
15. Mackay, R.: Report on Work Done at Darr es Salaam During the Period January, 1932-January, 1934. Abst. Trop. Dis. Bull., **33**:225 (March) 1936.
16. Daleas, P.: Bull. Soc. med.-chir. de l'Indochine, **13**:432-437 (May) 1935.
17. de Buck, A.; and Swellengrebel, N. H.: Proc. Roy. Acad., Amsterdam, **38**:452-454, 1935.
18. de Buck, A.; and Swellengrebel, N. H.: Proc. Roy. Acad., Amsterdam, **38**:335-343, 1935.
19. Trensz, F.: Bull. Soc. path. exot., **28**:558-560 (July) 1935.
20. Prudhomme, R. O.: Compt. Rend. Soc. de Biol., **119**:1115-1117, 1935.
21. Sydenstricker, V. P.; and Vryonis, G. P.: Jour. Lab. & Clin. Med., **20**:1094-1096 (July) 1935.
22. Pawan, J. L.: Ann. Trop. Med. & Parasit., **29**:327-328 (Oct.) 1935.
23. Kehar, N. D.: Rec. Mal. Surv. India, **5**:393-404 (Dec.) 1935.
24. Kehar, N. D.: Rec. Mal. Surv. India, **5**:405-411 (Dec.) 1935.
25. Helfferich, W. M. G.: Geneesk. Tijdschr. v. Nederl.-India, **74**:1438-1446 (Oct.) 1934.
26. de Buck, A.: Arch. f. Schiffs-u. Tropen-Hyg., **39**:342-345 (Aug.) 1935.
27. Cassuto, N.: Riv. di Malariologia (sez. I), **14**:384-390, 1935.
28. Putoni, V.: Riv. di Malariologia (sez. I), **13**:721-733, 1934.
29. Wilson, T.: Malayan Med. Jour., **10**:39-48 (June) 1935.
30. Lutrario, A.: Bull. Office internat. d'hyg. pub., **27**:1141-1160 (June) 1935.

CONSIDERATION OF THE MALARIA PROBLEM IN THE TENNESSEE VALLEY*

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We know malaria not as a single entity but as a problem of multiple aspects. It is not one but several diseases; not simple, but extremely complex in its epidemiology. The purpose of this discussion is, however, to outline some of the very broad considerations related to the regional aspects of control and factors, including that of administration, which may influence the prevalence of malaria for good or ill.

It is, of course, perfectly clear that the primary factor for consideration in analysis of malaria transmission must be that of water and its control. Without breeding surfaces and a food supply for anopheline mosquitoes, the index would quickly decline to the vanishing point. This condition would, however, be attended by such maladjustments in agriculture and food supply for other forms of life as to make the accomplishment in malaria prevention of no consequence. There would be no human beings to profit by it. Obviously, therefore, in regional programs of water control we must discover measures that produce maximum economic values with the minimum of adverse conditions to affect our well-being.

The valley of the Tennessee River and its tributaries is a vast watershed in which more rain falls each year than on any other region of similar size in the United States. Within the area of some 40,000 square miles live about two and one-half million people, and as a region it possesses an infinite variety of varying conditions relating to topography, climate, soil formation, and other elements of economic geography. It must be apparent that this rainfall and these widely varied conditions of land slope, climate, and soil possess vast potentialities for both good and evil. If properly related to a program of land use and conservation, the possibilities for economic welfare and protection of generations yet to come are vastly increased. If wasteful practices are the rule, the present resources through

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the destructive influences of water running riot and through exploitation of the land will soon be spent. The region can then but inadequately, if at all, support its population.

In earlier days comparatively little of the rainfall left the land by direct runoff. The larger fraction was held in the soil, by reason of its forest and ground cover, to be utilized in growing vegetation or to reach the streams through springs and ground water channels. Now the situation has been radically changed. Most of the rainfall reaches the streams by a rapid surface runoff, wasting itself so far as value to vegetation is concerned, and depleting the soil over which it passes in its haste to reach a stream channel. The result is too little water for crops and too little soil in which to grow them. It has been said, for example, that cultivated land in this region loses from 5 to 120 tons of soil to the acre each year. Obviously, the resultant silting of minor stream channels is another hazard and the vicious cycle there becomes complete.

This change in the way the water leaves the land has apparently had only one beneficent result to redeem its many destructive effects. Malaria has declined, not only in the Valley, but for the same reason of lessened breeding surfaces, the whole section of the United States east of the Mississippi River and certain sections immediately west of it show a lower mortality than in earlier years. A map of malaria mortality in 1870 would indicate a considerable ratio of deaths extending up into the region of the Great Lakes and up the Atlantic coastal plain to the latitude of Philadelphia or New York. Now, this cause of death has retreated to a position south of the Ohio River to include parts of eastern Texas, Louisiana, Arkansas and southeast Missouri.

The problem of the public health agency and the conservationist, simply stated, is to reproduce as far as possible the natural water control of earlier days without reproducing the malaria hazards of those days, for reduction of these hazards through the quickened runoff of water over the surface of the soil spends too freely of our basic resources and may eventually add to the malaria hazard in low-lying areas.

Economically, such a process resembles burning a home to rid it of rodent infestation. Conversely, of course, refusal to recognize the factors concerned with malaria prevention is comparable to the fireman who drowns the family in his anxiety to protect property from a fire.

Thus, to those of us in the public health field this program of water control in the Tennessee Valley becomes of direct and paramount importance; regardless of whether we serve in local, state, or Federal health organizations. We need to study its elements and extent and to define developing physical readjustments in terms of their impact upon the population. Granting the basic necessity of water control from a social and economic point of view, we must convert potential liabilities into actual assets by intelligently directed procedure undertaken as a team with a common objective.

Let us, therefore, briefly examine the principal aspects of this water control program which is just now the most important single "consideration" of the "malaria problem" in the Tennessee Valley. We may arrange them in two broad categories, as follows:

(1) Artificial storage in rivers that are publicly owned. Here we must consider the physical and economic requirements of water storage such as dams, reservoirs, stream flow, and rainfall; navigation, river facilities, and related transportation problems; and electricity as a by-product of these measures.

(2) Natural storage on land privately owned. It has been said that if we could hold on and in the soil of the region only 10 inches of the average annual rainfall of from 40 to 80 inches, we would keep back more water than can be contained behind two Norris Dams. Hence this group of measures is concerned with the agricultural requirements for natural water storage, including farm management, sods, legumes, forests, plant foods, and plant food production, terracing of slopes, and the various other measures entering into control of the so-called "Little Waters" in relation to the land.

The importance of considering the whole of the problem, rather than of isolating a single phase of it such as malaria control, is readily apparent from study of this grouping of measures for the reasons, first, that we must not convert portions of our country into "biological deserts," and, second, the naturalistic approach to the control of mosquito breeding appears to offer the best possibility of finding effective measures that are within the economic range of existing resources. Thus we must take into account not only the aspect of the problem with which we as public health workers are directly concerned, but also interests of biologists and others concerned with the conservation of natural resources and preservation of balance in other forms of life. We must constantly weigh one value against another in order to ascertain the relative worth of one procedure as compared to another. When land is drained, the result in mosquito control must be weighed against loss of useful life and lowering of ground water level. When water is impounded, it must be in such manner as not to increase the hazard of malaria. When reservoirs are cleared and prepared for an impounded pool, it must be done in such manner as to conserve useful growths and at the same time to minimize floatage. In the operation of reservoirs the circumstances conducive to wild life protection must be considered as well as conditions required for mosquito control. In short, we must view the whole picture and arrive at a coordinated program of work which, to the greatest possible degree, comprehends all related elements and considers the whole problem of conservation rather than a disarticulated fragment. It is clear that human values must take precedence over other values, but let us accurately determine the true human values. There is need for the control of disease, but there is also need for more productive lands, for forestry programs, for wild life conservation, and for ground water held through water-retaining crops which are such essential factors to improved agricultural economy. In brief, there is need to consider the whole range of vital and physical economics, in which malaria prevention is one purpose, and to make each purpose serve each other purpose as completely as lies within present possibilities. We may summarize the varied inter-relationships and their basic value to each other as follows:

(1) Storage of water in the water table, thus hold-

ing it in the land, has the primary values of (a) preventing soil erosion, (b) nourishing crops, (c) maintaining stream levels, (d) promoting an even flow of power, (e) saving the costs of expensive structures for artificial storage (dams), and (f) making no mosquito breeding places.

(2) By contrast, failure to utilize natural storage of water in the land (a) provides mosquito breeding places, (b) displaces water holding spaces, (c) hinders navigation, and (d) weakens the whole vital and physical economy of the region.

(3) The means to the end of natural water control and storage are found in (a) land use changes, (b) mechanical structures, (c) thickened ground cover (vegetation) by the use of phosphates, and (d) the incorporation of vegetable matter in the soils.

It is perfectly clear when we examine these elementary correlations that the prevention of malaria is not exclusively the concern of the public health agencies. On the contrary, it is so completely tied into the whole program of water control and so vitally related to the economic structure of the region that it must be the concern of every agency and every unit of organization having to do with community or regional development. It is our business to bring such agencies face to face with this responsibility and to recognize their broad purposes in order that the greatest good may come through a program each element of which is properly integrated with each other element.

Coming now to somewhat more specific details, it may be of interest to review the extent of the situation which artificial storage of water is producing and the steps which the Tennessee Valley Authority is taking to prevent an increased transmission of malaria. In the first place, this disease is or can become endemic throughout practically the whole extent of the Tennessee Valley region as is shown both by past history and more recent experience. The parasite of tertian malaria is the most prevalent, though that of estivo-autumnal persists to a very considerable extent. Indeed, recent years have shown a relative increase in infection with this parasite which, though data on which to base regional increases are lacking, is nevertheless indicative of the seriousness of the general situation because of the higher case fatality in estivo-autumnal malaria. In many sections the anopheline index has been below the threshold of danger for so long that the disease has practically disappeared, but experience a few years ago in a small Tennessee city shows that, with anopheline production at a level sufficiently high, transmission will assume epidemic proportions in a short time. Moreover, experience on the Norris pool shows how delicate is the balance between a high rate of mosquito production and controlled production, for with but slight elevation of the pool in the summer of 1935 profuse production was the result; whereas the year previous no breeding of *Anopheles quadrimaculatus* could be discovered despite a very careful survey of the entire area. Other studies indicate the range of bloods positive for malaria parasites to vary from zero to as high as 60 per cent of the persons examined, specimens having been taken at random by collection from every fifth family. Again, experience indicates that production rises enormously in a constant pool unless radical control measures are instituted, and an electricity transmission line path cut through the timber of a swamp may result in intensive breeding where sunlight reaches the water. The Norris pool in East Tennessee will have a shoreline of 705 miles at normal pool elevation, while that of Lake Wilson extends 130 miles and Lake Wheeler 1,130 miles, a total of 1,965 miles for pools now or soon to be impounded. When pools are impounded on the lower river during years to come, the impoundage will extend entirely across the western portion of Tennessee and through north Alabama. These facts are mentioned to emphasize the extent of the undertaking and to illustrate the need for realistic consideration. Of course, some existing problems will be simplified, for many areas now quite troublesome will be flooded out, and new areas will be carefully prepared for the impoundage. Nevertheless the situation is and will continue to be a challenge to every health agency working in the Valley, be it Federal, state, or local, for there is an opportunity not only to hold malaria transmission to present levels but ultimately to reduce its prevalence to a point where there is no longer a problem of serious consequence.

Since it will serve to illustrate both present and future opportunities, let me review the progress already made in planning for the future and in establishing organization and relationship for the present.

In any large regional program for water storage where malaria can occur, the control of mosquito production must be built into the structures and operating schedules of the water control facilities. With this in mind, the design of each dam constructed by the Tennessee Valley Authority provides a malaria "surcharge" permitting the seasonal and periodic fluctuation of water level necessary to the maintenance of clean shorelines and of other conditions inhibiting anopheline production. This element of design is agreed upon in consultation with engineers and medical officers of the health and safety division. In reservoir preparation an engineer of the division collaborates with the engineers of the reservoir clearance division in planning and supervising clearance operations. When preparation first begins, an engineer is assigned as resident sanitary engineer for the reservoir, and under supervision of a district consultant continues in that capacity during the stage of preparation and into the operating stages.

The impoundage schedule is agreed upon in a conference of the interested divisions of the Authority, including the health and safety division, and no reservoirs are filled during the mosquito breeding season. When the stage of operation is reached, fluctuation schedules within the malaria "surcharge" are arranged through coordinated load dispatching in such manner as to provide maximum values for mosquito control.

Simple enough in expression, these procedures are extremely complex in their relations to engineering requirements, power potentialities, and other aspects of the Authority's work. Hence this part of the program has required long study and very detailed planning by practically every division concerned with construction, engineering, operation, and health; a fact which is perfectly obvious when it is realized that the schedule

of operation must be designed to provide both the essentials to the control of mosquito production and optimum results in flood control and navigation.

Projects concerned with forestry and wild life conservation are cleared through a planning council and an inter-divisional committee upon which both the divisions having an interest are represented. This simple mechanism serves to coordinate requirements for the conservation of wild life and requirements for malaria control in such manner as to make each serve the ends of the other insofar as this is possible and to harmonize such differences in program as may develop. The following statement from a recent memorandum received by the Director of Health from the Director of the Forestry Division is quoted as an illustration of the manner in which the arrangement functions:

"* * * it is my understanding that the Health and Safety Division and the Forestry Division are in full accord with the program of joint cooperative work, tying in reforestation along the lake margins, the uses of cypress on shallow waters, including water areas behind dikes, the control over malaria mosquitoes everywhere, and especially the joint studies and research of the various biologic, aquatic and other problems calling for cooperative studies. The Forestry Division and the Health and Safety Division jointly approve of the program of a maximum of protection along the shores of the lake, coupled with a maximum of good forest management, good game management, the development of a maximum of good fishing, and with a minimum of mosquitoes. It is furthermore recognized by the Forestry Division that in all matters dealing with health, the necessities and considerations of the Health and Safety Division have priority."

Within the health and safety division both the health section and the sanitation section are concerned with planning and with field operations. The health section is primarily responsible for epidemiological study, health education, and technical supervision other than engineering. The sanitation section is responsible for field supervision and administration of control procedures and for engineering studies, both responsibilities being coordinated in the field with the work of the health section. There are available from the health section a medical malariologist; a biologist, especially qualified in entomology and limnology; and a specialist in health education. In the sanitation section a field supervising engineer trained in impounded water control procedures works with and through resident field engineers and technical aides. A small laboratory equipped for work in certain of the natural sciences as well as the usual diagnostic routines serves both sections. Thus the staff, though small, brings to bear upon both control and research problems a balanced and comprehensive technical training not always available in such operations.

The development of organization and procedure has extended beyond the division and even beyond the Authority, for relationships have been formed with the state health organizations, with the United States Public Health Service, and with the United States Department of Agriculture. A consulting board, consisting of five authorities in the field of malaria work, has been formally created from officers of the Public Health Service, the United States Department of Agriculture, and the Rockefeller Foundation. Technical planning and critical review of study and control procedures are functions of this board, both individually and collectively, for, though meetings of the entire group are held at intervals, individuals of the group are called into consultation whenever special problems arise.

Additional consultation and cooperation is fundamental to the achievement of successful results. The problem must be attacked along the whole broad front by biology, malariology, and engineering, and on the flanks by agriculture, economics, and education. Health agencies, educational organizations and institutions, agricultural agencies all must pool their efforts for the common good; hence the need for study and control procedure extends well beyond the immediate jurisdiction and operations of the Authority. There can and must be a second line of defense afforded by Federal, state, and local health services if full potentialities are to be realized. In recognition of these facts, contractual relations have already been established with the state health departments of Tennessee and Alabama under which the local health services around the reservoirs are strengthened and the community malaria control program extended through plans jointly formed and mutually agreed upon. The arrangement has been a particularly happy one. Through it responsibility is shared, a partnership of service is developed, and the whole structure for protection of the public health is strengthened. There is perhaps no other single arrangement which so completely illustrates the opportunity of working together. Moreover, it demonstrates a basic policy of the health and safety division, which is that full use must be made of existing facilities before new organization is created, and that work must be done as completely as possible with and through existing agencies.

In order that common purposes may be best served, members of the staff of the health and safety division are continuously available for such aid as they may render through the state health organizations to local health agencies. Especially valuable in this connection is an associate in health education who has, through cooperative work with local health departments already done much in the development of all important community "malaria consciousness."

We who are responsible in this complicated matter of malaria control have before us a job demanding the best in each and every one of us, of each element of organization, of each individual. Some of us are directly, others only indirectly, responsible. Some perceive part, others little of the broad implications in this job which lies beyond the direct service of health agencies. A few may have what appear to be conflicting interests and objectives. Others fail to see the chance for integration of effort, and still others may fail in comprehension of the scope of relationships. Still others may occasionally assume antagonistic attitudes because of the lack of opportunity for an interchange of views and information. These gaps must be closed. We can and we must develop that "team play" and interchange of service which will best enable us to achieve our common objective—the prevention of mortality and morbidity from malaria.

EXPERIENCES WITH MINOR DRAINAGE IN RELATION TO MALARIA RATES IN SOME MISSISSIPPI DELTA COUNTIES*

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It is planned to present in this paper the possible relationship between the malaria mortality rates in the Mississippi Delta and the cumulative amount of minor drainage executed. It is impossible to draw definite conclusions regarding this relationship, due to the fact that so many diverse factors have a direct or indirect effect on the malaria rate.

The Delta region of Mississippi is known as the Yazoo Basin of the Mississippi flood plain. In size it is approximately 65 by 180 miles and contains more than four and one-half million acres. It is bounded on the west by the Mississippi River and on the east by the bluffs of the coastal plain, at the foot of which is the Yazoo River and its tributaries. The Yazoo River empties into the Mississippi River at Vicksburg and affords the only outlet for the waters of this region. The region is a flat plain, slightly inclined to the south and east with a slope of about 0.7 foot per mile.

The entire basin is traversed by a network of many streams and bayous whose margins are slightly higher than the general elevation of the intervening stretches of plain. This is the result of gradual deposition of silt during overflows. The present levee system was begun about 1882. The levee extends along the entire western boundary of this region and, except for occasional breaks, has afforded protection from overflows to all of this region except the southern part, which now probably suffers more from backwater than formerly.

The development of this territory was given great impetus by the construction of levees and the installation of major drainage systems. As a result of these major improvements, there was an extensive immigration into this area from the non-malarious hill sections. Due to the above mentioned facts, this region was very favorable to the propagation of malaria.

The major drainage in the Delta was inaugurated about 25 to 30 years ago and most of this work was completed before 1928. Only a very few small projects have been completed since that time, and none since 1930. These large drainage canals were primarily constructed to reclaim, for agricultural purposes, the vast swamps of this region. This work was laid out and executed by engineers who had no conception of the fundamental principles of malaria control. No attempt was made to drain the ponds and swamps left by this major drainage.

In April, 1929, a program for malaria control for the Delta region of Mississippi was inaugurated by Doctor Mark F. Boyd. This program was designed to make use of every accepted method for the control of malaria, and placed special emphasis on the execution of minor drainage. These minor drainage activities were designed to take up where the major drainage work had left off, in order to eliminate breeding areas of anophelines in proximity to towns, cities, and thickly settled communities. It was believed that besides the direct results that would be obtained, it would indirectly affect the malaria rates by: first, stimulating interest in a general drainage program, particularly on farms; and, second, demonstrating to the State Highway Department and county officials the value of a properly constructed and maintained system of ditches.

In those counties where the major drainage had largely been completed, the promotion of minor drainage offered a means of eliminating remaining depressions (ponds and swamps) which produced anophelines.

Boyd, after an extensive survey of the region, said,

"Very nearly any retained, natural water will produce *quadrimaculatus* in the Delta. In our opinion, the result of any efficient drainage accomplished in the Delta will be satisfactory from a malaria control standpoint, even though the primary motive has been agricultural reclamation."

In order to study the relation between the amount of drainage that has been executed and the malaria death rate, the Delta counties have been divided into groups based on the amount of minor drainage executed. Conditions in the three groups to be studied are nearly comparable, except for the amount of drainage done in each.

Group 1* consists of four counties in which the most minor drainage has been executed; Group 2* consists of four counties in which less minor drainage has been done; and Group 3* consists of three counties in which practically no minor drainage has been accomplished.

Each of the counties in Groups 1 and 2 is served by a full-time county health department, while none of those in Group 3 has such health service. The counties in Groups 1 and 3 are adjacent, while those in Group 2 are scattered but are adjacent to those in Groups 1 and 3. All counties in these three groups lie wholly within and comprise the Delta.

Tables 1, 2, and *3* show the malaria mortality rates per 100,000 population for each county of the respective groups for the years 1920-1935. *Chart 1* shows these rates and trends for each group. These rates are accepted as a more trustworthy guide than are morbidity rates.

A study of the rates for Mississippi reveals a cyclic trend of about five to six years' duration. These cycles are noted in every region and group of counties.

A study of the three groups under discussion shows that Group 3 has the highest rate throughout the past 16 years; Group 2 has the second highest; and Group 1 the lowest. The trend line of Group 1 declines at a

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*Group 1 consists of Bolivar, Humphreys, Sunflower, and Washington Counties.

Group 2 consists of Coahoma, Issaquena, Leflore, and Sharkey Counties.

Group 3 consists of Quitman, Tallahatchie, and Tunica Counties.

slightly more rapid rate than does that of Group 3, while that of Group 2 declines at a much slower rate than either of the Groups 1 or 3.

A further study of these tables reveals the following facts, which are presented in *Table 4*. During the 16-year period, the percentage of reduction in the malaria rates was 68 for Group 1, 60 for Group 2, and 48 for Group 3. During the period 1920-1929, prior to the inauguration of minor drainage, the average rates for Groups 1, 2, and 3 were 56.7, 67.5, and 90.7, respectively. During this period, the rates decreased 79 per cent in Group 1, as compared with 63 per cent in Group 2, and 46 per cent in Group 3.

During the 6-year period, 1930-1935, after the inauguration of minor drainage, the average rates were, for Group 1, 34.0; for Group 2, 47.4; and for Group 3, 62.7; showing an increase in all groups for this period. The increase for Group 1 was 31 per cent; for Group 2, 55 per cent; and for Group 3, 35 per cent.

The minor drainage activities in Mississippi from 1929 to 1935 are given by years for each group in *Table 5*. The total amount of these activities for each group during this period is presented in *Table 6*. *Chart 2* is a graphic presentation of *Table 7*, which shows the number of acres drained and the malaria rates by years for each of the groups from 1928 to 1935. The figures listed under "acres drained" are cumulative; that is, the number of acres for each particular year represents the total number of acres drained to date in that group.

A study of these tables shows that 183 miles of ditches constructed under this program drained 10,834

Table 1

MALARIA MORTALITY RATES IN GROUP 1
(Per 100,000 Population)

Year	Bolivar	Humphreys	Sunflower	Washington	Group
1920	90.87	159.27	114.04	58.53	95.24
1921	82.17	159.96	113.58	58.10	92.51
1922	95.18	116.79	109.24	55.89	90.45
1923	89.96	80.59	75.16	51.73	74.17
1924	47.20	41.59	32.62	19.04	34.73
1925	49.33	90.16	45.51	37.86	49.74
1926	33.24	83.60	35.54	24.46	37.29
1927	56.31	51.57	40.96	24.31	42.87
1928	52.34	33.59	53.97	26.08	32.95
1929	21.40	12.31	23.02	9.24	17.80
1930	29.41	20.08	26.90	12.86	23.43
1931	15.12	15.72	14.52	10.96	13.98
1932	18.91	26.93	21.18	30.89	23.46
1933	61.07	79.15	60.46	37.94	57.40
1934	54.80	55.40	56.20	53.89	55.10
1935	33.35	39.82	26.08	28.58	30.64

Table 2

MALARIA MORTALITY RATES IN GROUP 2
(Per 100,000 Population)

Year	Coahoma	Issaquena	Leflore	Sharkey	Group
1920	103.00	199.30	113.01	91.71	112.31
1921	111.34	95.28	98.40	127.25	107.41
1922	84.35	83.71	80.06	99.18	84.61
1923	60.26	42.93	51.40	56.80	55.13
1924	66.50	14.68	31.53	35.57	45.01
1925	81.68	30.16	45.67	71.29	62.32
1926	67.35	46.51	27.33	92.88	52.42
1927	57.77	63.78	36.62	50.12	56.82
1928	94.57	32.83	43.37	50.23	63.66
1929	32.65	33.82	43.96	14.38	35.57
1930	43.10	.00	22.26	21.61	29.18
1931	27.73	17.43	21.62	21.61	23.77
1932	44.36	52.31	43.80	50.44	45.15
1933	69.04	69.75	85.24	86.47	78.53
1934	66.30	87.19	58.10	57.64	62.44
1935	38.99	34.87	48.52	57.64	45.32

Table 3

MALARIA MORTALITY RATES IN GROUP 3
(Per 100,000 Population)

Year	Quitman	Tallahatchie	Tunica	Group
1920	208.67	75.13	73.43	109.82
1921	208.19	122.57	199.86	166.09
1922	80.26	114.35	111.64	104.32
1923	133.59	94.90	111.17	119.94
1924	40.47	33.53	86.27	49.49
1925	74.68	69.92	28.75	60.47
1926	60.11	61.60	52.50	58.78
1927	104.96	58.86	114.07	86.92
1928	119.12	61.72	113.61	92.46
1929	56.28	47.30	80.14	58.76
1930	31.49	30.61	75.11	42.54
1931	53.99	22.49	46.75	53.19
1932	41.58	30.92	69.85	44.31
1933	96.36	84.34	139.14	99.86
1934	69.06	75.91	97.00	79.07
1935	46.36	56.23	73.61	57.41

Table 4

PERCENTAGES AND AVERAGE RATES OF INCREASE AND DECREASE IN MALARIA

Period	GROUP 1			GROUP 2			GROUP 3		
	Average Rate	Per Cent Decrease	Per Cent Increase	Average Rate	Per Cent Decrease	Per Cent Increase	Average Rate	Per Cent Decrease	Per Cent Increase
1920-1929	56.7	—79		67.5	—63		90.7	—46	
1930-1935	34.0		+31	47.4		+55	62.7		+35
1920-1935		—68			—60			—48	

acres in Group 1, while 90 miles in Group 2 drained 3,355 acres. In Group 3, nearly 4 miles of ditches were constructed, draining 91 acres. The amount of work done was fairly uniform for each year throughout the entire period of operation.

DISCUSSION

The mortality rate for Group 1 has been lower than that for Group 3 for the entire period from 1920 to 1935. This rate was greater than the rate for Group 2 from 1922 to 1924, and then remained under Group 2 for the rest of the period. The rate of increase in this group from 1930 to 1935 was less rapid than in either Group 2 or 3. It is interesting to note that Bass, in his studies of malaria in 1916 in the largest and most populous county of Group 1, stated that it was "the most heavily infected locality in the State of Mississippi."

During the past seven years, the Division of Malaria Control has drained many sloughs, ponds, and swamps in the Delta. These had an area of more than 14,000 acres and were located within the corporate limits or in flight range of towns, cities, and thickly settled communities.

Filling has proven the most popular and effective method of elimination of anopheline breeding areas in the minor drainage activities. This work has eliminated more than 10 acres of ponded water breeding anophelines within the corporate limits and has affected the entire population of 20 towns and cities. The primary reason for its popularity has been that it is permanent, it has eliminated the cost of anti-larval control, and it has reduced the hauling cost of city garbage and trash.

Many other factors have had a direct effect on the malaria rate in at least three of the counties of Group 1. In two counties, intensive malaria control studies and therapeutic control were inaugurated by Bass in 1914. From 1929 to 1931, a comprehensive anti-larval program was conducted by H. E. Hingst, now of the U. S. Public Health Service, in four towns of one county in Group 1. Three of these counties were settled early; hence, much of the pioneer work incident to clearing land and draining swamps was done before reclamation work was begun in some of

Table 5

MINOR DRAINAGE ACTIVITIES IN MISSISSIPPI, 1929-1935

GROUP 1

Activity	1929	1930	1931	1932	1933	1934	1935
Length new ditch	8,205	73,472	76,464	42,112	94,315	95,932	65,174
Length rechanneled	5,000	50,565	63,097	156,772	138,580	66,978	31,852
Total length put to grade	13,205	124,037	139,561	198,884	232,895	162,910	97,026
Number acres drained	157	1,400	1,974	2,587	2,837	1,721	158
Number ponds filled	2	1	1	0	0	42	42
Length tile installed	4,625	2,866	0	0	0	0	0

GROUP 2

Activity	1929	1930	1931	1932	1933	1934	1935
Length new ditch	27,077	62,138	35,164	13,455	20,448	54,968	2,630
Length rechanneled	3,000	21,998	58,110	88,952	35,460	49,419	3,620
Total length put to grade	30,077	84,136	93,274	102,407	55,908	104,387	6,250
Number acres drained	401	255	722	690	571	716	0
Number ponds filled	0	0	1	0	0	47	0
Length tile installed	0	490	0	0	0	0	0

GROUP 3

Activity	1929	1930	1931	1932	1933	1934	1935
Length new ditch	0	0	0	1,200	0	1,600	7,380
Length rechanneled	0	0	0	0	0	6,480	3,704
Total length put to grade	0	0	0	1,200	0	8,080	11,084
Number acres drained	0	0	0	75	0	10	6
Number ponds filled	0	0	0	0	0	0	6
Length tile installed	0	0	0	0	0	0	30

the newer Delta counties. These counties in Group 1 also undertook major drainage development at an earlier date than the other Delta counties.

Recently, a public health worker stated that agencies sponsoring conservation projects are increasing malaria faster than malaria workers are reducing it. This may be true in some sections; however, in Mississippi, practically all of the recently impounded lakes are located

Table 6

MINOR DRAINAGE ACTIVITIES IN MISSISSIPPI, 1929-1935

TOTALS

Activity	Group 1	Group 2	Group 3
No. miles new ditch	86.30	40.88	1.92
No. miles rechanneled	97.12	49.34	1.92
No. miles put to grade	183.42	90.22	3.84
No. acres drained	10.834.00	3.355.00	91.00
No. ponds filled	88.00	48.00	6.00
No. feet tile installed	7,491.00	490.00	30.00

Table 7

MALARIA MORTALITY RATES AND ACRES DRAINED BY MINOR DRAINAGE*

Year	Group 1		Group 2		Group 3	
	Acres Drained	Malaria Rate	Acres Drained	Malaria Rate	Acres Drained	Malaria Rate
1928	0	32.95	0	63.66	0	92.46
1929	157	17.80	401	35.57	0	58.76
1930	1,557	23.43	656	29.18	0	42.54
1931	3,531	13.98	1,378	23.77	0	53.19
1932	6,118	23.46	2,068	45.15	75	44.31
1933	8,955	57.40	2,639	78.53	75	99.86
1934	10,676	55.10	3,355	62.44	85	79.07
1935	10,834	30.64	3,355	45.32	91	57.41

*The figures showing the acres drained are accumulative.

Chart 1

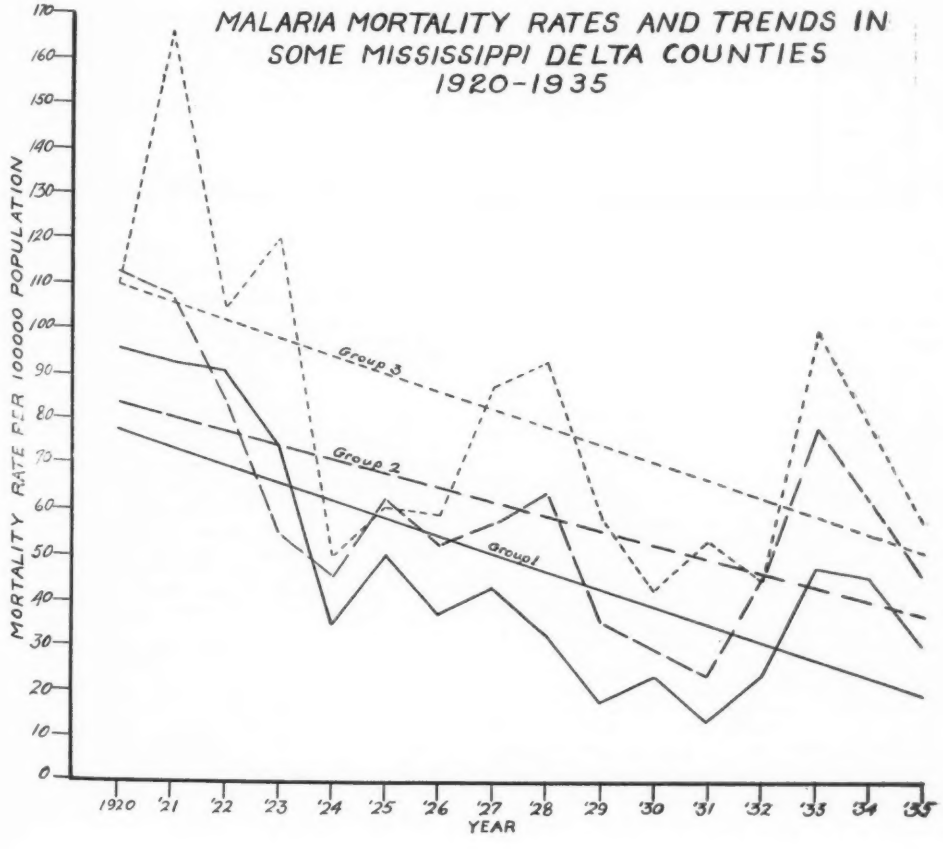
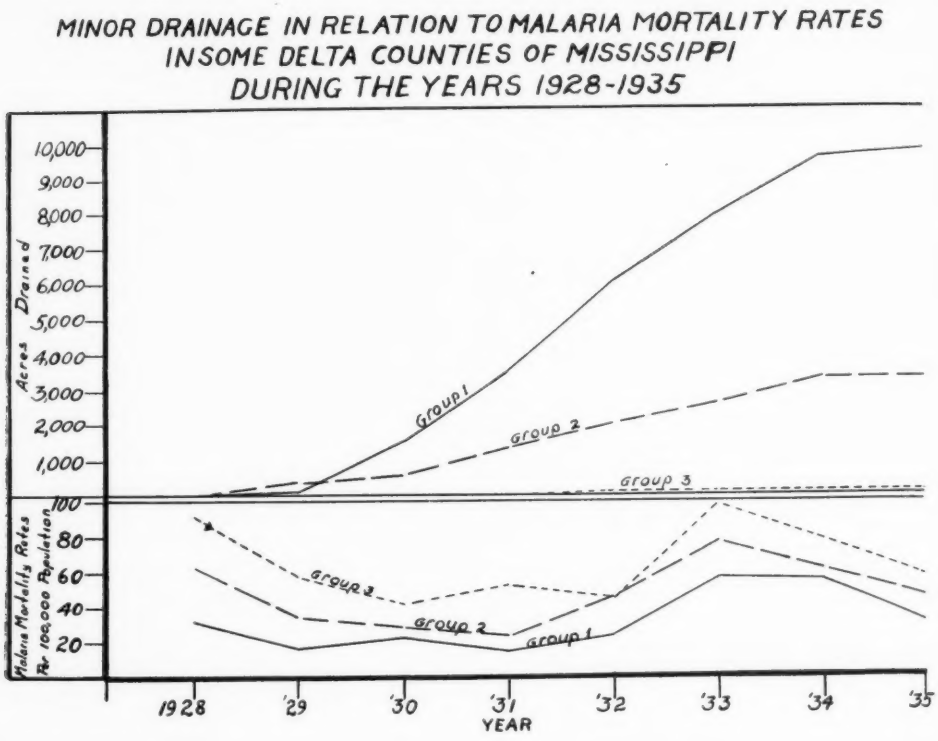


Chart 2



in sparsely settled areas; whereas, malaria control drainage is confined to urban areas affecting large populations.

After a thorough study of the above data, the conclusion is reached that it is most difficult to evaluate the work that has been executed in the Delta section of Mississippi, and that it is unwise to conclude that certain factors have been the most important in reducing the incidence of this disease. The data obtainable are meager and the many factors affecting the increase or decrease of the disease are so numerous and interlocked as to make positive statements impossible.

It is possible, however, to visualize the indirect benefits accruing from the educational work started in 1929. One tangible result of this educational work has been the definite improvement in the quality of drainage being executed by planters, surveyors, practicing engineers, and county and state highway officials. Properly constructed ditches are being executed now on plantations and in towns and highways; whereas, eight or nine years ago the same engineers and organizations created malaria hazards. These hazards were constructed of borrow pits that were difficult to drain and culverts built above grade.

One of the most important indirect benefits and one that will have far reaching effect is the change in attitude since 1931 of the Mississippi State Highway Department. This department now requires complete drainage of borrow pits on all new highways.

CONCLUSIONS

While an attempt is made to present the amount of minor drainage in relation to the amount of malaria, as determined by the mortality rates, no attempt is made to draw definite conclusions as to the degree of benefit received from this work.

In addition to any benefit directly gained from the minor drainage program, the benefit derived from the additional drainage done by individuals and organizations, and the great improvement in the quality of this work has been worth many times the cost of the program.

LePrince, Gorgas, and others proved in Panama and Cuba that malaria can be controlled, and this fact was further demonstrated in army cantonments during the World War when the case rate was only one-half of one per cent of that during the Spanish-American War.

This tremendous loss caused by malaria in the Southern States must be stopped, and it can be if adequately trained personnel are financed and permitted to utilize the known methods of control.

Southern Medical Journal

JOURNAL OF THE

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C. P. LORANZ, Secretary, Treasurer and General Manager

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EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Thirty-First Annual Meeting
New Orleans, Louisiana, November 30-December 1-2-3, 1937

KIDNEYS AND PARATHYROIDS

If the numerous endocrine products isolated in the last fifteen years have been a little slower than was hoped in relieving the symptoms of arthritis, hyper- and hypotension, nephritis and schizophrenia, or in combating infection, they have yet tremendously broadened the fields of physiology and clinical diagnosis.

Parathyroid effects have been particularly instructive because they may be followed by rather simple blood chemical studies. Typical parathyroid adenoma is a clear-cut and rare condition, yet clinical disease of these glands should be common, for several reasons. The hormone is closely associated with metabolism of calcium and phosphorus, which in turn are the chief constituents of bone and are the disordered elements of rickets and of the various other disturbances of bone formation. The hormone may influence body economy in many ways, and in turn may be stimulated by other organs or probably by the calcium and phosphorus intake.

Calcium is a basic element and phosphorus an acid one in the body. The parathyroids are capable of increasing the excretion of phosphorus or calcium and lowering the blood level of the one and raising that of the other. They must, therefore, be intimately concerned in the maintenance of the neutrality of the blood, a function of vital importance in body economy. The glands themselves in turn are regulated by the amount of blood phosphorus, according to Boston workers,[1] and hyperplasia may be produced by phosphorus injections. Kidney insufficiency interferes with the excretion of phosphorus, which thus accumulating in the blood stream could cause a compensatory parathyroid hypertrophy. Thus the glands which normally stimulate excretion of phosphorus, in the presence of too much of the same element, may enlarge from overwork.

Clinically, the parathyroid tumor has been thoroughly studied and its diagnostic signs rather definitely outlined.[2] In this condition the serum calcium is high and the phosphorus low: there is an increased excretion of both calcium and phosphorus in the urine and these elements are apparently being wasted by the body. There are also characteristic changes in the roentgen picture of bones. Albright[1] and his associates have described a new syndrome of human parathyroid disease, in which there is a high inorganic phosphorus level in the blood serum with evidence of phosphorus retention in the body. There is a slight reduction of the serum calcium, a marked acidosis, and numerous calcium deposits may be seen in the neighborhood of joints. There is extreme calcification of the media of the arteries, osteitis fibrosa of all bones, and enormous enlargement of all parathyroid tissue. It is suggested that the etiologic factor of this condition lies in the kidneys, and that this is the adult counterpart of renal rickets.

In primary hyperparathyroidism, the Boston investigators suggest, when the parathyroids initiate the disease, the serum calcium should be high. It should be normal or low in parathyroid disease which originates in the kidney, like that described above.

Further necropsy studies will be of interest, of the parathyroids in kidney diseases and in cases of chronic phosphorus poisoning.

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1. Albright, Fuller; Drake, T. G.; and Sulkowitch, H. W.: Renal Osteitis Fibrosa Cystica: Report of a Case with Discussion of the Metabolic Aspects. Bull. Johns Hopk. Hosp., **60**:377 (June) 1937.

2. Henson, R. R.; Shorr, E.; McClellan, W. S.; and Dubois, E. L.: A Case of Oteitis Fibrosa Cystica. Jour. Clin. Investig., **8**:215, 1930. Bauer, W.; Albright, F.; and Aub, J. C.: Idem, p. 229. Albright, F.; Aub, J. C.; and Bauer, W.: Hyperparathyroidism. J.A.M.A., p. 1276, 1934. Editorial, Parathyroid Disease. Sou. Med. Jour., **27**:819, 1934.

RINGWORM IN THE UNITED STATES

One of the disorders of modern life about which many papers have been written in the past ten years, is the spread of fungus diseases of the skin, particularly tinea of the feet. Swimming pools and public showers are usually blamed for the latter condition, and these have steadily increased in number. Ringworm once established is difficult to eradicate, though its symptoms may be readily controlled. The infected individual is apt to remain a permanent carrier.

Several rather simple methods of prevention have been described and publicized, and sufficient time has elapsed for these to have influenced the incidence of the disease. There have been a number of careful and complete analyses of health records, of large groups of college students particularly, since health services have become general in schools and universities. Pels and Schlenger,[1] of Baltimore, in 1932, reported an incidence of 60 per cent in a group of adults examined in this Eastern city. Present day studies are of particular interest, to determine whether the recommended prophylactic measures have influenced the occurrence of the infection, whether there are any signs of decrease.

Alderson and Reich,[2] of Leland Stanford University, have published the most recent report on skin diseases among students. In the men's health service of Stanford in the eight-year period between 1928 and 1936, they note, 5,354 new diagnoses of skin diseases were made. The ten most common conditions were listed by the authors. First and most frequent was tinea, which made up one-fourth of all dermatologic diagnoses during that period. Second was pyoderma; third, poison oak, since the plant is common in the woods about the University grounds. Acne, the California authors say, affects about half the members of the student body, though only 3.7 per cent sought treatment for it. Urticaria and pediculosis pubis were counted among the ten commonest skin diseases.

One-half of the students who entered the university already had some form of tinea of the feet. At the end of the first semester, the incidence had increased to 70 per cent: that is, very nearly three out of every four students were infected. The authors note that Pollitzer reported tinea as eighth or ninth in frequency among dermatologic patients observed in the years from 1873 to 1916, a considerably earlier period. The statistics are hardly comparable, but tinea of the feet has probably spread faster in this century than any other contagious disease, is rivaled in incidence only by the common cold, and from the report from the Pacific Coast, there is not the slightest evidence of any sort of let-up.

It would appear that the human being, like the fruit tree, may require spraying at stated intervals for control of prevalent micro-organisms, without waiting for symptoms or diagnoses. The United States Public Health Service is doing a beautiful piece of educational work in the control of syphilis and the venereal diseases, which soon should be reflected in venereal morbidity rates. The campaign for eradication of the social diseases is so promising that one is tempted to wish that the next mass attack might be turned on this milder but more widespread condition. The lowly tinea might serve as a worthy and also responsive public health objective.

1. Pels, I. R.; and Schlenger, Leo: Incidence of Dermophytosis of the Feet, with Comments on the Use of Trichophytin. Sou. Med. Jour., **25**:1066, 1932.

2. Alderson, H. E.; and Reich, A.: Incidence of Dermatosis in a Student Health Service. Arch. Dermat. & Syphilol., **36**:57 (July) 1937.

GLEANINGS FROM RECENT JOURNALS

Discoloration by Drugs.—One of the disadvantages of the use of "atebrin" for treatment of malaria is the yellow color that it often gives the patient. This tinting is temporary and disappears when the administration is stopped. A disturbing effect of prolonged use of silver salts is the occasional development of argyria, in which the patient turns a peculiar silver gray, and this condition is apparently permanent. Mercury is extensively used as an antisyphilitic and disinfectant. It is occasionally noted to cause darkening of the nails after luetic therapy and in persons who frequently rinse the hands in bichloride solution as in surgical or bacteriological work. The gray color cannot be washed off, but goes through the nail, where it remains till the nail has grown out. The new nail is normal. According to Callaway,[1] of the University of Pennsylvania, the same discoloration may be produced in nail pairings and in the horns of cattle after application of 5 per cent ammoniated mercury and subsequent exposure to light. Nails kept in the dark do not turn gray. Sulphur is abundant in the nails, and Callaway reports that the color is due to the reaction of

1. Callaway, J. Lamar: Transient Discoloration of the Nails Due to Mercuric Chloride. Arch. Dermat. & Syph., **36**:62 (July) 1937.

mercury with it to form the black mercuric sulfide.

Brain Potential.—Electric conductivity studies of various organs are being extended and would appear capable of giving useful diagnostic signs. It was shown last year by Yale investigators[1] that there is a sudden release of electric energy when small animals ovulate, and that the time of ovulation may thus be detected through the intact abdominal wall. The reactions of the human brain may be studied by means of an instrument known as the electroencephalograph, which resembles the electrocardiograph in principle. It is connected with the intact skull of the individual instead of with the heart, and a voltage is said to be obtained which is about one-hundredth that obtained in the electrocardiograph. Tracings of the normal brain have been studied, and of the brain under the influence of drugs, and during loss of consciousness in *petit mal* and from other causes. The pattern of a normal waking person, like his signature or his finger print, is said to be characteristic, and the records of identical twins are similar.[2] The instrument offers many possibilities for neurologic and psychiatric study.

Skin Softener.—There would appear to be a rapidly increasing number of papers on cosmetic subjects, and of cosmetic advertisements, in medical journals. These are subjects of considerable importance which were formerly somewhat ignored. Goodman,[2] of New York City, who has written a book on cosmetics, reports a new skin softening and cleansing agent, triethanolamine, which he says readily removes black heads, grease and the scaliness of seborrhea.

1. Burr, H. S.; Hill, R. T.; and Allen, Edgar: Detection of Ovulation in the Intact Rabbit. Proc. Soc. Exper. Biol. & Med., **3**:109 (Oct.) 1935. Editorial, Sou. Med. Jour., **29**:89, 1936.

2. Gibbs, F. A.; Gibbs, E. S.; and Lennox, W. G.: Arch. Int. Med., **60**:154 (July) 1937.

3. Goodman, Herman: Cosmetic Dermatology. Triethanolamine. Arch. Dermat. & Syph., **36**:116 (July) 1937.

TWENTY-FIVE YEARS AGO

From Journals of 1912

Medical Education in America.[4]—"The Carnegie Foundation for the Advancement of Teaching has just issued its second bulletin on medical education, this time dealing with conditions found in Europe. * * * This is the only civilized country which does not require of every medical student a preliminary education in physics, chemistry and biology either prior to or at the beginning of the medical course. * * * 'scandals in medical education exist in America alone.' * * * Well trained young physicians in Germany find no difficulty in attaching themselves to the retinue of hospital staff physicians and surgeons and thus procuring for themselves active scientific work, while in America this is practically impossible * * * in Europe the trustees of hospitals freely open their wards to students. * * * Although the determined movement for better medical education in this country began only about eight years ago, astonishing progress has been made since that time. Our vast oversupply of inferior medical schools is rapidly being reduced. Where there were 166 medical schools in the United States in 1904—nearly half of the world's supply—now there are only 118."

Indications for Hysterotomy.[1]—"I wish especially to urge that placenta praevia must be considered an indication for hysterotomy which is to be given the preference over any other method of delivery. In the toxemias of pregnancy * * * it is to be seriously considered as a rival of less radical measures."

Anoci-association in Surgery.[2]—* * * "Harmful or nocuous associations are called 'noci-associations.' If, then, an operation be so planned that all harmful or nocuous associations are prevented, this state is designated 'anoci-association,' that is to say, 'without noci-association' * * * the management of a patient up to the point of anesthesia should be by only those nurses, orderlies and physicians who are humanitarian psychologists; the anesthetist should be even more of a psychologist, and preferably a woman, for somehow the world has an instinctive confidence in the ultimate good intentions of woman as compared with those of man. * * * Anoci-association may further be promoted by the use of pleasant nitrous oxid instead of the irritating, suffocating ether.* * * Then when under anesthesia, the brain may be entirely isolated from the field of operation by as careful and as complete infiltration into the latter of a solution of 1:400 novocain as if no general anesthesia had been given. * * * At the close of the operation, if the zone of operation is cut off for a considerable time from communication with the brain by an injection of quinine and urea hydrochloride, after-pains and postoperative nerve-exhausting stimulation are avoided.* * * The mortality rate, especially in abdominal operations, * * * has been more than cut in two. Anoci-association takes from surgery most of its proverbial harshness, and on this principle we can now actually perform shockless operations."

1. Deaver, John B.: Hysterotomy. J.A.M.A., **59**:90, 1912.

2. Crile, George W.: The Results of Operations, Especially Abdominal, Performed on the Principles of Anoci-association. J.A.M.A., **59**:114, 1912.

4. Medical Education in Europe. Editorial, J.A.M.A., **59**:40, 1912.

New Orleans—Where We Meet

RECREATION IN NEW ORLEANS*

It is hardly unusual to hear references made to the historic associations of New Orleans. The whole wide world knows of the Vieux Carré, preserved practically intact for the past century and a half, with its antiques and many historical recollections. Every high school graduate who ever studied American history is familiar with the establishment of the city by Bienville, and the numerous obstacles he had to overcome; the purchase of the Louisiana Territory from Emperor Napoleon of France by President Jefferson; the Battle of New Orleans, in which Andrew Jackson won an overwhelming victory over the British under General Pakenham; and the Union occupation of the city by General Benjamin Butler in 1862. Many glamorous episodes of the past find associations in New Orleans.

But an important phase of New Orleans history, too often overlooked, is its role in the development of sport and recreation in this country. Oldest festival of the city, in which New Orleans annually teaches the rest of the world how to play, is Mardi Gras, begun in 1827. It is held in late February or early March, but its gay spirit is characteristic of the city all year round. Then in 1837, the first organized race course in the country was opened in New Orleans, where Audubon Park's most beautiful lawns and lagoons are today, and a later race course occupied the site of the beautiful Metairie Cemetery. The year 1849 saw the organization of the present Southern Yacht Club, second oldest boating club in the United States, and baseball was introduced here in the late 1860's. The New Orleans Lawn Tennis Club was established in 1876, when tennis was still a "sissy game," and is the oldest tennis club in the country. John L. Sullivan beat Paddy Ryan for the heavyweight championship of the world near New Orleans in 1882, and three world's championship matches were once fought on successive nights in the old New Orleans Olympic Club. The first gloved contest, replacing the more brutal bare-knuckle battles, was fought in St. Bernard Parish, just over the Orleans Parish line. Football entered Louisiana in 1892, and 1893 saw the beginning of the Tulane-Louisiana State rivalry which continues to this day. Today, one of the South's leading sport events and nation's football classics is the annual Sugar Bowl Game, played January 1 in the Crescent City.

Thus did the history of sport unfold in the South, with New Orleans consistently leading in its making. And there was sufficient reason for the city's premier position. For one thing, it was the largest city in the South. For another, it was very cosmopolitan. For a third, it had large, spacious parks, with adequate facilities provided for outdoor sports, along with other natural advantages. And perhaps most important of all, its balmy, temperate climate, with a year round average temperature of 69 degrees, provided conditions ideal for outdoor sport and recreation. New Orleans thus had a head start on the rest of the country which it has not yet relinquished.

Members of the Southern Medical Association, meeting there November 30-December 1, 2, 3, will find no lack of recreational facilities for their enjoyment. Whether interested only as a spectator, or in active participation in sports, in driving and touring, or only in lazy relaxation, there is something to fill the bill.

For the spectator, the city offers horse-racing, at the Fair Grounds of the Louisiana Jockey Club, or attendance at some of the various other sports events. Boxing and wrestling matches are scheduled weekly, and the annual gridiron grudge battle between Tulane and Louisiana State Universities will be held in New Orleans November 27, just three days before the convention opens. Although no date is yet selected, possibilities are that a Southern Yacht Club regatta will be held during this period.

For the active participants, facilities are provided for every type of outdoor sport, and there is not even any necessity of leaving the city limits. New Orleans, unique in this as in all other things, provides hunting and fishing ground within the confines of the city. The Louisiana hunting season customarily begins early in November and lasts about ten weeks, and nowhere is such an abundant supply of game and wild life found as in Louisiana during this period. Especially abundant are migratory fowl, and huntsmen customarily

A view of old and new New Orleans. In the foreground are the iron trellised balconies so typical of the old homes and old buildings in the French Quarter. In the background can be seen a few of the skyscrapers that modern New Orleans has built.

*Prepared for the Southern Medical Journal by the Publicity Department of the New Orleans Association of Commerce.

A view in City Park, originally a 1,382-acre plantation. In this park can be seen some of the finest specimens of live oaks in America, sturdy, massive old trees, over a hundred years old, with long beards of Spanish moss drooping from their branches to the ground like long, silvery swords. There are also more than twenty varieties of palms in the park.

bag the limit on ducks, geese, woodcock and snipe. But rabbit, deer, and even bear, all legitimate prey to the hunter, are present in sufficient quantity to please anyone. The Pass A La Loutre Hunting Club is nationally famous. It is state owned and operated, with 66,000 acres of the world's finest hunting grounds. All hunting necessities can be provided.

Fishing, of course, is a year round sport in New Orleans, with Lake Pontchartrain and numerous small lakes all in the vicinity, the excellent fishing ground of Chef Menteur and the Rigolets within city limits, and the farther fields of the Gulf and Mississippi Coast within easy driving distance. Tarpon, redfish, speckled trout, black bass (called "green trout" locally), croakers, drum, sheepshead, red snappers, flounders, Spanish mackerel, bluefish and pompano are highly esteemed Louisiana fish.

For the golf enthusiast, there are three public 18-hole courses, one in Audubon Park and two in City Park, not to mention several private courses connected with the country clubs. For the tennis lover, Audubon Park and City Park together offer approximately 50 courts, about half of which are equipped for night play. The player has his choice of brick, clay and hard-surfaced courts. Private clubs here too add to the facilities.

Both public parks provide rowing and fishing facilities, in artificially constructed, annually-stocked lagoons. Yachting goes on all year round on Lake Pontchartrain, at the famous Southern Yacht Club. Audubon Park is equipped with a fine stable and approximately five miles of bridle paths where equestrians can take a quiet canter. The path leads through some of the most beautiful sections of the park.

Tours in and about the city introduce the visitor to many new and interesting sights. Within the city itself there is the endless novelty and Old World charm of the Vieux Carré, the natural beauty of the parks, the hustle and bustle of the riverfront, the unruffled calm or squally tempestuousness of Lake Pontchartrain, the quaintness of the Garden District with its spacious gardens and antebellum homes, the scholarly serenity of the universities, and the reverential atmosphere of the many-tombed cemeteries. But an even broader field awaits the visitor who has time to investigate the surrounding territory.

On the river, below the city, is the old plantation of Chalmette de Ligny, where the Battle of New Orleans was fought. The site is marked by a giant marble obelisk, and the famous Versailles Oaks, finest grove of live oaks on the face of the earth. Above the city one meets successively two of the most monumental engineering feats of our time, the Mississippi River Bridge and the Bonnet Carré Spillway. The former, built at a cost of $13,000,000, is a toll-free outlet to the West for railroad, vehicular and pedestrian traffic. The latter is a flood control project, where at high water stages Mississippi flood waters can be diverted out through Lake Pontchartrain.

Most appealing of the short trips is to the West, out the Old Spanish Trail, to the Evangeline Country, with its shady bayous, moss-draped oaks, old French settlements and Cajun inhabitants. Out the Old Spanish Trail to the east, one encounters the Mississippi Gulf Coast with its famed winter resorts.

Driving up the river, on an airline concrete highway, one reaches Baton Rouge, where the State Capitol building and Louisiana State University are located. The L. S. U. Medical Center, however, is situated in New Orleans.

South of the city, across the river, is the drive to Lafitte, a small town named after the blustering pirate, Jean Lafitte, and still farther down is Barataria Bay, where the pirates had headquarters, an intricate maze of bayous and embayments, many of which are still uncharted. Grand Isle, at the very edge of the Gulf, is a famous resort, especially noted for its fine fishing and surf bathing.

Book Reviews

Medical Urology. By Irvin S. Koll, B.S., M.D., F.A.C.S., Attending Urologist, Michael Reese Hospital. 431 pages, illustrated. St. Louis: The C. V. Mosby Company, 1937. Cloth $5.00.

Parts of this volume are worthy of recommendation, others are deficient and severely to be criticized.

The list of references following each chapter will be helpful to those who wish to review the literature. Unfortunately, however, they are not always brought up to date.

The anatomy, bacteriology, etiology, pathology, symptomatology, and diagnosis are well covered. The treatment advised is from the author's personal experience, and it fails in many instances to coincide with the standard treatment of today.

The chapters dealing with specific and non-specific

infections in the lower urinary tract in the male and female contain many worthy suggestions.

The greatest criticism is of the part pertaining to prostatism and kidney infections. The advice to delay operations on the prostate until a complete retention occurs is not in accord with the present trend of thought. In the treatment of infections of the kidney, no mention is made of the ketogenic diet or mandelic acid therapy. Both of these have passed through the experimental stage, have demonstrated their superiority in a large percentage of kidney infections, and should be included in any text on medical urology.

The illustrations are not up to the present day standard.

A Manual of Radiological Diagnosis for Students and General Practitioners. By Ivan C. C. Tchaperoff, M.A., M.D., D.M.R.E. (Camb.), Assistant Radiologist and Radium Registrar, St. Thomas's Hospital, London. 256 pages, illustrated. Baltimore: William Wood & Company, 1937. Cloth $6.00.

This book, which is intended as a synopsis of the essentials of radiological diagnosis, was written primarily for students and the general practitioner. The first chapter deals briefly with x-ray physics; the second and third chapters with the osseous system, in general and regionally. These 126 pages with 175 illustrations comprise approximately one half the volume. The illustrations are very clear and well labeled, but the descriptions are brief and at times confusing; for example, what the Americans refer to as giant cell tumor, the author calls myeloma of the bone.

The other half of the volume contains brief chapters on the chest; gastro-intestinal tract; gallbladder, kidney and urinary tract; the female generative system; tumors of the spinal canal and ventriculography. All these chapters are much too brief to furnish an adequate source of information to the radiological student or to the qualified radiologist. To the undergraduate student and general practitioner, as an aid to the understanding of the expert's diagnosis, it should be of service.

The Diagnosis and Treatment of Arthritis. By Russell L. Cecil, M.D., Sc.D., Professor of Clinical Medicine, Cornell University; Associate Visiting Physician, New York Hospital, New York City. Henry A. Christian, M.D., Sc.D., LL.D., General Editor of the Series. (Reprinted from Oxford Monographs on Diagnosis and Treatment). 263 pages, illustrated. New York: Oxford University Press, 1937. Cloth $4.75.

The writer is recognized as an authority on the subject of arthritis. After a brief survey of the generally accepted classification of joint diseases, there is a detailed presentation of the various groups of arthritis. Especially noteworthy are chapters on acute rheumatic fever, rheumatoid arthritis, and the arthritis of menopause. Modern treatment, including vaccines, antistreptococcic sera, foreign protein, hyperpyrexia, and physiotherapy, is clearly outlined. Although a brief manual, it is complete enough to present an excellent and authoritative survey of the age-old problem of arthritis and the accepted modern therapeutic measures.

Southern Medical News

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination and review of case histories of Group B applicants by the American Board of Obstetrics and Gynecology will be held in various cities in the United States and Canada on Saturday, November 6, 1937. The next general examination for all candidates (Groups A and B) will be held in San Franciso, California, on June 13 and 14, 1938, immediately prior to the American Medical Association meeting. Application blanks and booklets of information may be obtained from Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburg (6), Pennsylvania. Applications for these examinations must be filed in the Secretary's office not later than sixty days prior to the scheduled dates of examination.

ALABAMA

Dr. Albert C. Jackson, Jasper, has been elected President of the Alabama Hospital Association.

Deaths

Dr. James Ratchford Taylor, Anniston, aged 76, died March 12 of bronchopneumonia.

Dr. Rufus C. Jones, York, aged 78, died March 15 of pneumonia.

Dr. Gibson Reynolds, Montgomery, aged 60, died recently of heart disease.

Dr. Wiley Simeon Adams, Cherokee, aged 52, died in April.

Dr. Howell T. Heflin, Birmingham, aged 60, died June 18.

Dr. R. M. Buchanan, Decatur, died July 1.

ARKANSAS

The State Medical Board has elected the following officers for the coming year: Dr. W. A. Snodgrass, Little Rock, President; Dr. L. T. Evans, Batesville, Vice-President; Dr. L. J. Kosminsky, Texarkana, Secretary-Treasurer.

The Arkansas State Pediatric Association has elected the following officers for the coming year: Dr. Edwin C. McMullen, Pine Bluff, President; Dr. Garland D. Murphy, El Dorado, Vice-President; Dr. Madeline A. M. Melson, Little Rock, Secretary.

The Ninth Councilor District Medical Society has elected the following officers for the coming year: Dr. D. K. McCurry, Green Forest, President; Dr. H. V. Kirby, Harrison, Vice-President; Dr. J. H. Fowler, Harrison, Secretary-Treasurer.

The Baptist State Hospital, Little Rock, has elected the following officers for the coming year: Dr. Joe F. Shuffield, Chief; Dr. K. W. Cosgrove, Vice-Chief; Dr. T. Duel Brown, Secretary.

Dr. Ernest Stroud, Jonesboro, has been made President of the Jonesboro High School Alumni.

Dr. Fount Richardson, Fayetteville, was recently made Physician to the University of Arkansas.

Dr. R. T. Smith, Fort Smith, has been made Vice-President of the Arkansas Hospital Association.

Dr. Edward W. Pillstrom has been made Health Officer of Coal Hill.

Dr. B. C. Middleton, Texarkana, was recently made Health Officer of Miller County.

Dr. Robert H. Johnson, Clarksville, has been made Health Officer of Johnson County.

Dr. L. J. Kosminsky, Texarkana, was recently made District Commander of the Twelfth District of the American Legion.

Dr. F. J. Scully, Hot Springs National Park, has been made Grand Commander of the Arkansas Knights Templar.

Dr. H. Fay H. Jones, Little Rock, is doing special work in London, Paris and Rome.

Dr. Alfred Hathcock, Fayetteville, has been taking postgraduate work in New York and Rochester, Minnesota.

Dr. Pierre Redman, formerly of Fort Smith, announces his association with Dr. B. H. Hawkins, of Mena.

Dr. John McCollough Smith, Morrilton, and Miss Marie Angehr were married June 17.

Deaths

Dr. James A. Foltz, Fort Smith, aged 59, died May 22 of heart disease.

Dr. Pleasant E. Terry, Holly Grove, aged 63, died April 29 of a heart attack.

Dr. John Marion Hooper, Batesville, aged 67, died May 19 from injuries received in an automobile accident.

Dr. Robert Rodney Dale, Texarkana, aged 52, died May 10.

Continued on page 26

40 Principal Conditions May Cause Trouble Here

THE problem of diagnosis and differentiation assumes commanding proportions when the urinary tract is involved. But efficient use of the x-ray examination has contributed mightily to the present-day accuracy with which urinary disease may be diagnosed and classified.

Simple radiography will outline the kidneys, indicating their size, shape, and position. . . . Pyelography will disclose calculus, sarcoma, tuberculosis, alterations in the renal pelves; also kinks in the ureter or obstructions. . . . Retrograde urography will demonstrate the presence of tumors, traumatisms, diverticuli, foreign bodies. In differential diagnosis, radiography will usually place the blame where it belongs. . . . The facts that are thus disclosed will suggest proper therapeutic procedure.

For utmost assurance of accuracy in diagnosis, every sufferer from urinary disease is entitled to a thorough radiographic examination. Eastman Kodak Company, *Medical Division*, Rochester, N. Y.

RADIOGRAPHS PROVIDE DIAGNOSTIC FACTS

Continued from page 872

Dr. Joseph B. Shaw, Hot Springs National Park, aged 57, died May 28.

DISTRICT OF COLUMBIA

Commander Charles S. Stephenson, Medical Corps, U. S. Navy, has been made a member of the National Advisory Health Council to succeed Rear Admiral Charles S. Butler, resigned.

Dr. Harry F. Dowling, Director of Laboratories, George Washington University Hospital, received the merit prize of the Washington Medical and Surgical Society for research on pneumonia.

Dr. Charles W. Warren, formerly of Washington, has moved to Upperville, Virginia.

Dr. Joseph E. Gladstone, formerly of Washington, has moved to Exmore, Virginia.

Dr. Charles Hampton Mauzy, Jr., Washington, and Miss Elizabeth Anne Thaxton were married June 19.

Dr. Harold Wellington Jones, Washington, and Mrs. Mary Morrison Camper were married May 1.

Dr. Albert G. Love, Washington, and Miss Evelyn Ramsey were married May 4.

Deaths

Dr. Edgar Morris Watson, Washington, aged 25, died March 9 of pneumonia.

Dr. George M. Ruffin, Washington, aged 61, died May 29.

FLORIDA

The Florida State Board of Medical Examiners has elected the following officers for the coming year: Dr. John M. Mann, Lake Butler, President; Dr. Julius C. Davis, Quincy, Vice-President; Dr. William M. Rowlett, Tampa, Secretary.

Dr. Edward M. L'Engle, Jacksonville, has been made Registrar of the Bureau of Vital Statistics of the State Board of Health.

Dr. C. C. Witt, Arcadia, has been made Supervisor of District Number Two of the State Health Department.

Dr. Harold C. McDowell, formerly of Gastonia, North Carolina, has been made a member of the staff of the Florida Medical Center, Venice.

Dr. Rollin D. Thompson, formerly of Statesan, Wisconsin, has moved to Orlando, where he is in charge of the new State Tuberculosis Sanatorium.

Dr. Leonidas M. Anderson, Lake City, has been made Inspector of the State Institutions and Administration Advisor by the State Board of Health.

Dr. L. Y. Dyrenforth, Jacksonville, has been made a Fellow in the Florida Engineering Society.

Dr. Francis T. Holland, Tallahassee, has opened offices on Monroe Street.

Dr. Raymond Ralston, formerly of Pennsylvania, has moved to Lakeland, where he is associated with Dr. R. R. Sullivan.

Dr. William J. Buck, Belle Glade, and Miss Sue Florence were married May 22.

Dr. James L. Chalker and Miss Mary C. Fore, both of Ocala, were married April 14.

Deaths

Dr. Harriet Elizabeth MacSorley, Miami, aged 56, died March 17 of surgical shock following an operation for hip reconstruction.

Dr. Albert A. Dixon, Pensacola, aged 57, died April 28 of cerebral hemorrhage and arteriosclerosis.

Dr. Benjamin Perry Matheson, Fort Myers, aged 73, died March 31 of nephritis and arthritis.

Dr. Ira Elton Neer, Miami, aged 61, died March 30 of cirrhosis of the liver.

Dr. Bertha Frederica Carl Frommell, Orlando, aged 64, died March 13 of coronary thrombosis.

Dr. J. Frank Harris, Sarasota, aged 80, died March 28.

Dr. Richard Leffers, Lakeland, aged 59, died April 12.

Dr. Morris David Hartman, Homestead, aged 48, died April 13 of cerebral hemorrhage due to a head injury received in an automobile accident.

Dr. Frederick Wilhelm Schnauss, Jacksonville, aged 70, died April 20.

Dr. J. Knox Simpson, Jacksonville, aged 51, died May 19.

Continued on page 28

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Continued from page 26

GEORGIA

Dr. Edgar G. Ballenger, Atlanta, has been elected President-Elect of the American Urological Association.

Dr. Glenn J. Bridges, Atlanta, has been made Health Officer of Jenkins County to succeed Dr. Hugh B. Senn, Millen, resigned.

Dr. Thomas B. Phinizy, Augusta, has been made Commissioner of Public Health of Richmond County.

Dr. Glenville Giddings, Atlanta, has been made a member of the Board of Governors of the American College of Physicians.

Dr. Thomas W. Collier, College Park, has been made Health Officer of Dougherty County to succeed Dr. Hugo Robinson, resigned.

The Stephens County Medical Society has elected the following officers for the coming year: Dr. J. H. Terrell, Toccoa, President; Dr. E. F. Chaffin, Toccoa, Vice-President; Dr. C. L. Ayers, Toccoa, Secretary-Treasurer.

Dr. William C. Humphries, formerly of Griffin, has moved to Acworth.

Dr. Hubert H. Askew, Atlanta, has returned after taking postgraduate work in New York City.

DEATHS

Dr. Jeremiah Dumas Malone, Marietta, aged 76, died April 11.

Dr. Lewis McFarland Gaines, Atlanta, aged 59, died May 24 of coronary thrombosis.

Dr. Charles B. Almond, Winder, aged 66, died April 16.

Dr. Jefferson Davis, Toccoa, aged 70, died March 30.

Dr. John Oscar Elrod, Forsyth, aged 59, died April 21.

Dr. Zia Mabsut Bagdadi, Augusta, aged 54, died April 11 of coronary thrombosis.

Dr. Hinton James Eve, Augusta, aged 59, died April 27.

Dr. Nathaniel C. Goss, Ellijay, aged 75, died April 29.

Dr. Beverly Wood Yawn, Eastman, aged 51, died May 3.

Dr. James W. Jones, Thrift, aged 71, died May 18 from the effects of a black widow spider bite.

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Only graduates of medical schools who have completed a year of intern service will be admitted to the course.

The course begins October 11, 1937. Fee for the course is $600.00.

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KENTUCKY

The Kentucky State Medical Association will hold its annual meeting at Berea, September 5-7.

The Caldwell County Medical Society has elected the following officers for the coming year: Dr. B. K. Amos, Princeton, President; Dr. J. M. Dishman, Greensburg, Vice-President; Dr. W. L. Cash, Princeton, Secretary-Treasurer.

Dr. Gradie R. Towntree, Hickman, has been made Assistant Health Officer at Louisville.

Dr. Charles G. Baker, Louisville, has been made Health Officer of Fulton County.

Dr. Joseph J. Gerkins, Bedford, has been made Health Officer of Fleming County.

Dr. Chadwick W. Christine, Flemingsburg, has been made Health Officer of Jefferson County.

Dr. James C. Hancock, formerly of Fort Apache, Arizona, has been made Health Officer of Hickman.

DEATHS

Dr. Ruce Marion Miller, Ivyton, aged 78, died March 22.

Dr. Richard L. Foster, Danville, aged 66, died April 27 of angina pectoris.

Dr. John Franklin Crawford, Clay, aged 59, died April 17 of cerebral hemorrhage.

Dr. C. E. Leatherman, Louisville, aged 73, died April 15 of heart disease.

Dr. Edward Merriett Watkins, Sacramento, aged 80, died March 30.

Dr. William Francis Grubbs, Hazel, aged 71, died April 2 of coronary thrombosis.

Dr. Aubrey Vernon Jones, Louisville, aged 56, died April 5 of heart disease.

Dr. Joseph G. Gray, Franklin, aged 60, died April 13.

LOUISIANA

The Lafayette Parish Medical Society has elected the following officers for the coming year: Dr. A. R. Trahan, Lafayette, President; Dr. F. H. Davis, Lafayette, Vice-President; Dr. R. S. Hernandez, Duson, Secretary-Treasurer.

The St. Landry Parish Medical Society has elected the following officers for the coming year: Dr. S. J. Rozas, Opelousas, President; Dr. L. Lazaro, Opelousas, Vice-President; Dr. O. J. Bienvenu, Opelousas, Secretary-Treasurer.

The Vermilion Parish Medical Society has elected the following officers for the coming year: Dr. J. T. Abshire, Kaplan, President; Dr. Leo Saporito, Kaplan, Vice-President; Dr. A. A. Comeaux, Abbeville, Secretary-Treasurer.

The Louisiana State Gynecological Society has elected the following officers for the coming year: Dr. Edward L. King, New Orleans, President; Dr. Armand G. McHenry, Monroe, Vice-President; Dr. Henry B. Alsobrook, New Orleans, Secretary.

The Louisiana State Pediatric Society has elected the following officers for the coming year: Dr. Clarence H. Webb, Shreveport, President; Dr. Ralph J. Talbot, Monroe, Vice-President; Dr. Ruth G. Aleman, New Orleans, Secretary-Treasurer.

The Louisiana Society of Gastro-Enterology has elected the following officers for the coming year: Dr. Abraham L. Levin, New Orleans, President; Dr. Herbert L. Weinberger, New Orleans, Vice-President; Dr. Louis Ochs, Jr., New Orleans, Secretary.

Dr. F. L. Jaubert, New Orleans, has been made President of the Louisiana Society for Crippled Children.

Dr. Henry W. E. Walther, New Orleans, has been appointed Professor and Head of the Department of Urology at Louisiana State University in the Graduate School of Medicine.

Dr. Valentine H. Fuchs, New Orleans, has been made Professor of Otorhinolaryngology and Director of the Department at Louisiana State University in the Graduate School of Medicine.

Dr. Lambert O. Clark, Lafayette, was presented the Civic Award for 1936 for outstanding service to the community.

Dr. William L. Smith, formerly of New Orleans, has moved to Washington, D. C.

Dr. James Madison Batchelor, New Orleans, and Mrs. Bernice Bowling Passman were married May 1.

DEATHS

Dr. Joseph Shackelford Stephens, Natchitoches, aged 76, died March 27 of arteriosclerotic heart disease and nephritis.

Continued on page 30

ESTIMATED HYPOTHETICAL SALT ANALYSIS

(Grams per 100 c.c.)	Cow's Milk	Breast Milk	Similac
Calcium combined with Protein	.054 gm.	.024 gm.	.030 gm.
Mono-Magnesium Phosphates	.103 gm.	.027 gm.	.027 gm.
Di-Calcium Phosphates	.175 gm.	.000 gm.	.000 gm.
Di-Potassium Phosphates	.230 gm.	.000 gm.	.000 gm.
Potassium Citrates	.052 gm.	.103 gm.	.103 gm.
Sodium Citrates	.222 gm.	.055 gm.	.055 gm.
Calcium Citrates	.119 gm.	.059 gm.	.059 gm.
Iron	Trace	Trace	Trace
Sulphur with Protein	.022 gm.	.001 gm.	.001 gm.
Phosphorus with Protein	.022 gm.	.001 gm.	.001 gm.

C. W. Martin, M. D., New York State Journal of Medicine, September 1, 1932.

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Continued from page 28

Dr. Orville Jay Richardson, Manifest, aged 51, died March 29 of heart disease.

Dr. William L. Fisher, Lake Charles, aged 71, died March 7 of cerebral thrombosis.

Dr. LeRoy Cockfield, Bermuda, aged 52, died April 4 of myocarditis and diabetes mellitus.

Dr. M. David Haspel, New Orleans, aged 50, died June 26.

Dr. Russell Edward Stone, New Orleans, aged 59, died June 18 from complications following acute appendicitis.

MARYLAND

Dr. Magnus I. Gregersen, Professor of Physiology, University of Maryland School of Medicine, Baltimore, has been made Professor and Head of the Department of Physiology at Columbia University College of Physicians and Surgeons, New York.

Dr. James Eugene Hemphill, Baltimore, and Miss Mary Nancy Ray were married June 21.

Deaths

Dr. J. Henry Wishard, Leitersburg, aged 80, died March 27 of cerebral hemorrhage and bronchopneumonia.

Dr. Frederick Henry Charles, Cumberland, aged 75, died March 19 of arterial hypertension and myocarditis.

Dr. James Edward Bell, Baltimore, aged 46, died April 25 of lobar pneumonia.

MISSISSIPPI

The Marshall County Medical Society has elected the following officers for the coming year: Dr. C. R. Senter, Byhalia, President; Dr. Ira B. Segle, Holly Springs, Vice-President; Dr. Herbert Phillips, Holly Springs, Secretary-Treasurer.

Dr. J. S. McNeal, formerly of Crossville, has moved to Moorhead.

Dr. S. D. Newell, Inverness, Dr. George J. Mancill, Indianola, and Dr. J. W. Lusk, Ruleville, have been taking postgraduate work in New Orleans, Louisiana.

Dr. I. B. Bright, Greenwood, has been taking postgraduate work in New York.

Dr. Edwin M. Butler, Centreville, and Miss Redolia Davis were married May 5.

Deaths

Dr. William Willis Durden, Columbus, aged 92, died April 18 of hip fracture.

Dr. John F. McCarty, Ovett, aged 71, died in April of pneumonia.

Dr. Charles B. Watkins, Clinton, aged 70, died March 20.

Dr. Miles Jasper Wilhoit, Water Valley, aged 54, died recently.

Dr. W. M. Duke, Sunny Side, aged 56, died May 27.

MISSOURI

The Missouri State Medical Association has elected the following officers for the coming year: Dr. Bernard W. Hays, Jackson, President-Elect; Dr. Dudley S. Conley, Columbia, President; Dr. Edward J. Goodwin, St. Louis, Secretary.

The Missouri Public Health Association has elected the following officers for the coming year: Dr. Merl P. Moorn, Columbia, President-Elect; Dr. Edwin H. Schorer, Kansas City, President; Dr. John W. Williams, Jefferson City, Secretary.

Dr. Ralf Hanks, Fulton, has been made President of the Missouri Association for Mental Hygiene.

Dr. Max A. Goldstein, St. Louis, received the honorary degree of Doctor of Laws from Washington University.

Dr. Karl E. Maneval, Mexico, has been made Health Officer of Audrain County to succeed Dr. Robert W. Berrey, deceased.

Dr. Oliver S. Gilliland, Kansas City, has been made President of the Kansas City Eye, Ear, Nose and Throat Society.

Continued on page 32

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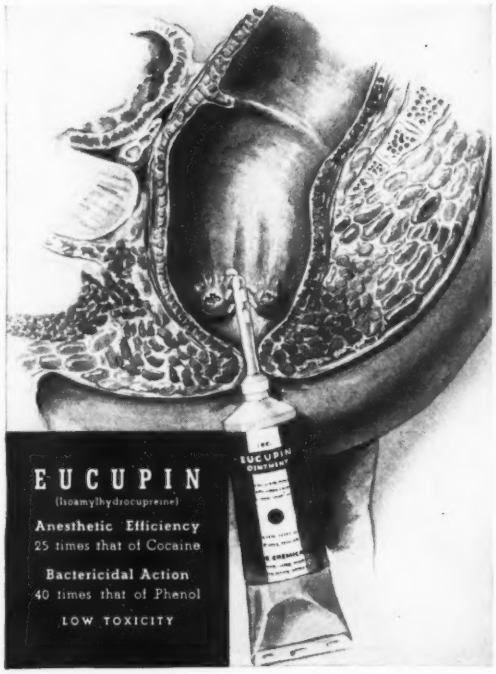

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Continued from page 30

The Missouri Society for the Advancement of Gastroenterology has elected the following officers for the coming year: Dr. Horace W. Soper, St. Louis, President; Dr. Frank D. Gorham, St. Louis, Vice-President; Dr. Charles W. Duden, St. Louis, Secretary; Dr. Lee Pettit Gay, St. Louis, Treasurer.

Dr. Philip A. Shaffer, Professor and Head of the Department of Biologic Chemistry at Washington University School of Medicine, has been made Dean of the School.

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Dr. Lee Wallace Dean, St. Louis, received the gold medal of the American Laryngological Association at its annual meeting in Atlantic City in June.

DEATHS

Dr. George A. Humbert, St. Louis, aged 75, died April 16 of chronic myocarditis and arteriosclerosis.

Dr. William Otto Winter, St. Louis, aged 67, died March 31 of heart disease.

Dr. Fredrick S. Markel, Allenville, aged 75, died March 16 of heart disease.

Dr. Lynn Gray Taylor, Kansas City, aged 77, died March 31 of thyrotoxicosis and arteriosclerosis.

Dr. Henry Davis Rothgeb, Willow Springs, aged 69, died March 29 of cerebral hemorrhage.

NORTH CAROLINA

The Medical Society of the State of North Carolina has elected the following officers for the coming year: Dr. James B. Sidbury, Wilmington, President-Elect; Dr. Wingate M. Johnson, Winston-Salem, President; Dr. Thomas W. M. Long, Roanoke Rapids, Secretary.

Dr. John W. Roy Norton, Raleigh, has been made Assistant Director of the Division of Preventive Medicine of the State Board of Health to succeed Dr. Thomas C. Worth.

The North Carolina Public Health Association has elected the following officers for the coming year: Dr. Malcolm T. Foster, Fayetteville, President; Dr. Joseph A. Morris, Oxford, Vice-President; Dr. Avon H. Elliot, Wilmington, Secretary.

Dr. Harry L. Johnson, Hickory, has been made President of the North Carolina Hospital Association.

Dr. Robert P. Noble, Raleigh, has been made President of the North Carolina Radiological Association.

Dr. Harold C. McDowell, formerly of Gastonia, has moved to Venice, Florida, where he is a member of the staff of the Florida Medical Center.

Dr. Paul A. Yoder, Winston-Salem, has been elected President of the North Carolina State Tuberculosis Association.

Dr. Wyman P. Starling, Clinton, formerly Health Officer of Sampson County, has resigned to enter private practice.

Dr. Warren Dallas Carter, Wilmington, has been made Health Officer of Burke and Caldwell Counties.

Dr. E. L. Kendig, formerly of Richmond, Virginia, has moved to Wilmington.

Dr. Cecil Collins Swann, Asheville, and Miss Dorothy Everett Spalding were married May 8.

Dr. Louis Carroll Robert and Miss Jessie Speight Ward, both of Durham, were married April 30.

Dr. William Blount Norment and Miss Katherine Williams, both of Greensboro, were married recently.

Dr. Herbert Junius Fox, Franklinville, and Dr. Frances Faison Hill, Durham, were married May 15.

DEATHS

Dr. Clarence Dupree Jones, Hillsboro, aged 70, died April 3 of angina pectoris.

Dr. Lee Johnson, Gastonia, aged 49, died April 8.

Dr. Thomas Patton Cheesborough, Asheville, aged 67, died

Continued on page 34

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Continued from page 32

April 3 of internal hemorrhage due to ruptured varicosities of the esophagus and cirrhosis of the liver.

Dr. John Ellis Ashcraft, Fayetteville, aged 76, died April 26 of heart disease.

Dr. Harris Hartwell Bass, Henderson, aged 59, died May 18.

Dr. N. W. Mackie, Yadkinville, aged 38, died May 21.

OKLAHOMA

Dr. Henry K. Speed, Sayre, was made President-Elect of the Oklahoma State Medical Association and Dr. Samuel A. McKeel, Ada, was installed as President at the recent meeting in Tulsa.

Dr. James L. Adams, Hobart, has been made Health Superintendent of Kiowa County.

Dr. Harry B. Hall, Boise City, has been made Health Superintendent of Cimarron County.

Dr. Robert M. Church, Stilwell, has been made Health Superintendent of Adair County.

Dr. J. T. Lowe, Mangum, has been made Health Superintendent of Greer County.

Dr. Glen W. McDonald, Ada, has been made Director of the County Health Unit in Pontotoc County.

Dr. John D. Campbell, formerly of Fairview, has moved to Buffalo, New York.

DEATHS

Dr. Arthur Herbert Haney, Oklahoma City, aged 41, died April 19 of pulmonary tuberculosis.

Dr. Oliver Bagby, Vinita, aged 78, died April 20.

Dr. Charles William Heitzman, Muskogee, aged 67, died March 8 of hypochromic anemia.

Dr. Myron Stephen Gregory, Oklahoma City, aged 67, died April 9 of coronary occlusion.

Dr. Jacob M. Stookbury, Shawnee, aged 68, died June 24 of a cerebral vascular accident.

Dr. John D. Leonard, Wagoner, aged 53, died April 24.

SOUTH CAROLINA

The Fifth District Medical Society has elected the following officers for the coming year: Dr. S. J. Blackmon, Kershaw, President; Dr. J. M. Settle, Great Falls, Vice-President; Dr. J. W. Brunson, Camden, Secretary.

Dr. Kirby D. Shealy, Columbia, and Miss Harriet Elizabeth Way were married recently.

DEATHS

Dr. Frank Duane Mower, Newberry, aged 57, died March 20 of cardiovascular renal disease.

Dr. Augustus Henry Hayden, Columbia, aged 77, died March 29 of arteriosclerosis and nephritis.

TENNESSEE

The Middle Tennessee Medical Society has elected the following officers for the coming year: Dr. C. M. Hamilton, Nashville, President; Dr. M. L. Hughes, Clarksville, Vice-President; Dr. D. W. Smith, Nashville, Secretary-Treasurer.

Dr. Howard Thompson Holden, Chattanooga, and Miss Dorothy Rodes were married in June.

DEATHS

Dr. James N. Baxter, Berlin, died in April.

Dr. John Kennerly Farris, Jr., Manchester, aged 70, died April 1 of gastric ulcer.

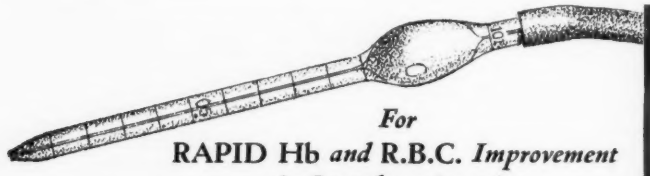
Dr. Green B. Gillespie, Covington, aged 92, died April 15 of myocarditis.

Dr. Joseph Vincent Power, Spencer, aged 64, died April 17 of pneumonia.

TEXAS

The State Board of Medical Examiners has elected the following officers for the coming year: Dr. O. B. Kiel, Wichita Falls,

Continued on page 36



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LIV-IRN Capsules allow administration of liver extract (its minerals and stimulating principles) and massive doses of iron in convenient form. They accomplish hematological recovery through rapid rise in hemoglobin percentage and red cell count . . . LIV-IRN Capsules are supplied in bottles of 84 and 500 capsules.

LIV-IRN Tablets are particularly suitable for children as they are easy to take. Co-ordinating ingredients achieve the desired change in the blood picture with a small number of tablets, in a relatively short time. Each tablet contains reduced iron, the secondary anemia fraction of fresh liver—plus irradiated yeast for stabilization of nervous, digestive and assimilating functions . . . LIV-IRN Tablets are available in bottles of 75 and 500 Tablets.

All LIV-IRN Products (including LIV-IRN Liquid) are chemically, biologically and clinically controlled; ethically produced and marketed; pleasant to take; always prepared from fresh U. S. government inspected livers.

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LARGER DOSAGE UNNECESSARY

Large doses of iron are no longer necessary for the successful treatment of hypochromic anemia. Independent investigations have proved that Hematinic Plastules yield rapid clinical response, equivalent to the results obtained from massive doses of other forms of iron • The average patient requires only three Hematinic Plastules Plain daily which obviates the unpleasant effects usually associated with massive iron feedings • We will be pleased to send samples and literature on request.

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Also for excursions, traveling, etc.

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answers many problems of hot weather diet:
1. For infants and growing children.
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3. For the sick and convalescent.
4. Whenever an easily prepared, highly nutritious food is desired.

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to be dissolved in the mouth are convenient to keep at hand, offering quick, sustaining nourishment at any time of the day or night. Especially useful while motoring, golfing, etc.

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Racine, Wisconsin.

Continued from page 34

President; Dr. W. C. Morrow, Gainesville, Vice-President; Dr. T. J. Crowe, Secretary-Treasurer.

The Texas Railway Surgeons' Association has elected the following officers for the coming year: Dr. J. H. Dorman, Dallas, President; Dr. A. L. Ridings, Sherman, First Vice-President; Dr. Joe Gilbert, Austin, Second Vice President; Dr. Ross Trigg, Fort Worth, Secretary-Treasurer.

The Texas State Heart Association has elected the following officers for the coming year: Dr. Edward H. Schwab, Galveston, President; Dr. M. D. Levy, Houston, Vice-President; Dr. Robert M. Barton, Dallas, Secretary-Treasurer.

The Texas Dermatological Association has elected the following officers for the coming year: Dr. Bedford Shelmire, Dallas, President; Dr. Everett R. Seale, Houston, Secretary.

The Texas Pediatric Society has elected the following officers for the coming year: Dr. John G. Young, Dallas, President; Dr. F. Otis Calaway, Houston, Vice-President; Dr. Frank H. Lancaster, Houston, Secretary-Treasurer.

The Texas Neurological Association has elected the following officers for the coming year: Dr. T. M. Dorbandt, San Antonio, President; Dr. J. C. Perry, Wichita Falls, First Vice-President; Dr. John A. McIntosh, San Antonio, Second Vice-President; Dr. Wilmer Allison, Fort Worth, Secretary-Treasurer.

Dr. K. W. Rowe, Kerens, has been made City Health Officer.

Dr. Charley Jones, Wellington, has been made City Health Officer.

Dr. John G. Young, Dallas, has been made Chief of the medical staff of the Richmond Freeman Memorial Hospital Clinic.

Dr. Robert S. Yancey, Dallas, has been made Chief Surgeon of the M. K. & T. Employes' Hospital Association.

Dr. J. M. Coleman, Plano, has been made Field Director of the Division of Maternal and Child Hygiene of the State Department of Health.

Dr. D. M. Rumph, Fort Worth, has been made Chairman of the Board of Directors of the City-County Hospital.

Dr. May Owen, Fort Worth, was given the Honorary Degree of Doctor of Science by the Texas Christian University.

Dr. Edgar H. Vaughn, Tyler, has been taking postgraduate work in Rochester, Minnesota.

Dr. W. P. Phillips, Greenville, has returned after taking postgraduate work in Chicago.

Dr. Jim M. Vaughn, Tyler, and Miss Bonna Bess Jones were married June 2.

Dr. Joe R. Wise, Fort Worth, and Miss Madge Brelsford were married May 17.

Dr. Robert Thomas Daniel, Fort Worth, and Miss Alberta Carl were married May 21.

Deaths

Dr. John Marquiss Blair, Houston, aged 78, died April 2.

Dr. William Prentice Farrington, Munday, aged 55, died April 17 of melanocarcinoma and cholecystitis.

Dr. Sterling O. Hays, Dallas, aged 67, died recently of coronary occlusion.

Dr. Walter Jackson Coleman, Mineola, aged 76, died March 28 of myocarditis.

Dr. Benjamin H. Bennett, Dallas, aged 74, died April 9.

Dr. Douglas Largen, San Antonio, aged 54, died April 16 of pneumonia.

Dr. Charles Massie Beavens, Port Arthur, aged 34, died recently.

Dr. Thomas E. Anderson, Carthage, aged 48, died May 6 of angina pectoris.

Dr. Garfield McCoy Hackler, aged 72, died May 6.

VIRGINIA

The Culpeper Medical Society has elected the following officers for the coming year: Dr. J. L. Stringfellow, Culpeper, President; Dr. Granville Eastham, Culpeper, Vice-President; Dr. O. K. Burnette, Culpeper, Secretary-Treasurer.

The Alleghany-Bath County Medical Society has elected the following officers for the coming year: Dr. Thomas M. Winn, Covington, President; Dr. R. L. Claterbaugh, Clifton Forge, Vice-President; Dr. R. P. Hawkins, Clifton Forge, Secretary-Treasurer.

The Virginia Society of Otolaryngology and Ophthalmology has elected the following officers for the coming year: Dr. Marshall H. Hood, Portsmouth, President; Dr. Charles T. St. Clair, Bluefield, Secretary.

Continued on page 38

Authoritatively Recognized

for PROMPT CURATIVE ACTION, TOLERANCE and LOW RELAPSE RATE

ATABRINE has received recognition not only in hundreds of published reports in medical journals throughout the world but also in a number of our standard textbooks on medicine and pharmacology. This constitutes a remarkable tribute to the important position which this relatively new drug occupies in the treatment of the various forms of malaria.

Atabrine has attained such prominence because of its manifold advantages over previous therapeutic agents. In the vast majority of cases, a short course with this drug produces quick extermination of the parasites, with prompt disappearance of the symptoms. Hence, as shown by numerous statistical studies, the possibility of relapses has been greatly reduced. Furthermore, Atabrine therapy has proved the most comfortable way of curing malaria.

Supplied in tablets of 0.1 Gm. (1½ grains), tubes of 15 and bottles of 100 and 500; 0.05 Gm. (¾ grain), bottles of 50 and 500. Ampules of 0.2 Gm., boxes of 5 (with distilled water).

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Continued from page 36

The Norfolk County Medical Society has elected the following officers for the coming year: Dr. Frank H. Redwood, Norfolk, President; Dr. William B. Newcomb, Norfolk, President-Elect; Dr. N. F. Rodman, Norfolk, Vice-President; Dr. Lockburn B. Scott, Norfolk, Secretary-Treasurer.

The University of Virginia Medical Society has elected the following officers for the coming year: Dr. Dudley C. Smith, President; Dr. Staige D. Blackford, Secretary-Treasurer.

Dr. L. Floyd Hobbs, formerly of Jewell Ridge, has moved to Alexandria.

Dr. E. T. Terrell, Williamsburg, has been made a member of the Board of Directors of the Williamsburg Lions Club.

Dr. Waldon M. Wattles, Farmville, has been made Resident Physician at the Southside Hospital.

Dr. Linwood Farley has been made Assistant Health Officer of the Valley Health District with headquarters in Luray.

Dr. J. M. Hurt, Blackston, has been made President of the Rotary Club of that city.

Dr. Claude C. Coleman, Richmond, has been made Clinical Professor of Neurological Surgery at the University of Virginia School of Medicine, Charlottesville.

Dr. Robert P. Stock, formerly of Stonega, has moved to Childersburg, Alabama.

Dr. M. H. Mund, formerly of Covington, has moved to Martinsville.

Dr. Walter E. Vermilya, formerly of Raleigh, North Carolina, has moved to Big Rock.

Dr. J. Bernard Jones, formerly of Towson, Maryland, has moved to Culpeper.

Dr. Charles W. Warren, formerly of Washington, D. C., has moved to Upperville.

Dr. Joseph E. Gladstone, Jr., formerly of Washington, D. C., has moved to Exmore.

Dr. E. W. Dodd, Jr., formerly of Roanoke, has moved to Gary, West Virginia.

Dr. James W. Tankard, formerly of Buffalo, New York, has moved to Franktown.

Dr. William McKenzie Bickers and Miss Virginia Fitzpatrick, both of Richmond, were married June 5.

Dr. Frederick Ralph Person, Williamsburg, and Miss Dorothy Italena Speese were married June 12.

Dr. John W. Devine, Jr., Lynchburg, and Miss Clara Roberts were married June 30.

Dr. Charles Allen Easley, Jr., Richmond, and Miss Elizabeth Fitzgerald were married recently.

Deaths

Dr. Frank Wandling Hornbaker, Occoquan, aged 62, died June 11.

Dr. Alonzo Leonidas Winfield, Richmond, aged 61, died June 22.

Dr. Samuel Downing, Newport News, aged 44, died June 14.

WEST VIRGINIA

The West Virginia State Medical Association has elected the following officers for the coming year: Dr. Charles W. Waddell, Fairmont, President; Dr. H. H. Haynes, Clarksburg, and Dr. A. A. Shawkey, Charleston, Vice-Presidents; Dr. T. M. Barber, Charleston, Treasurer.

Dr. Edward J. Van Liere, Morgantown, has been made Dean of the West Virginia University School of Medicine.

Dr. Harwood A. Taylor, Mullens, has been made Superintendent of the McKendree Emergency Hospital to succeed Dr. John N. Reeves, resigned.

Dr. James Beebe Hawes, Charleston, and Miss Helen Forbes White were married recently.

Deaths

Dr. Louis A. Bowling, Charleston, aged 76, died April 17 of pneumonia.

Dr. Herbert William David Garred, Charleston, aged 34, died March 12 of edema of the brain.

Dr. Frank T. McClintic, Marlinton, died April 15 of heart disease.

Dr. Walter Weldon Harloe, Matoka, aged 62, died April 17 of coronary occlusion.

Pancreas Therapy
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Oral Administration

An aid in the decreased capacity of the patient in states of pancreatic deficiency.

Bottles of 100, 500 and 1000 tablets.
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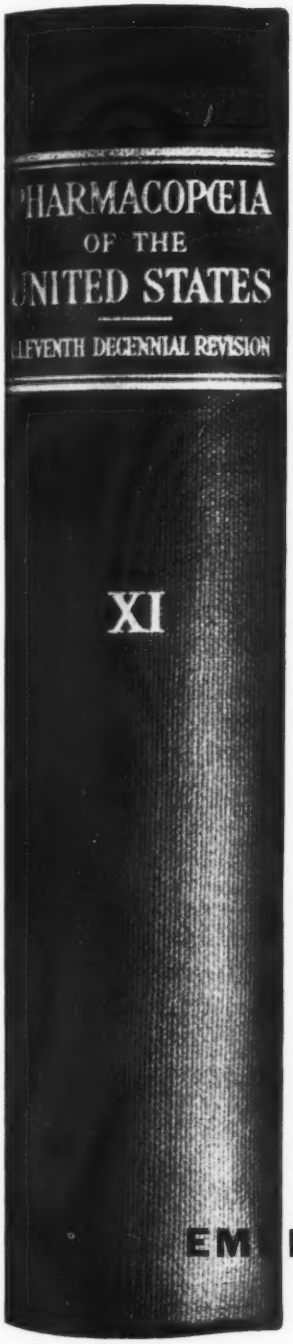

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Try this combination—for efficient analgesic action—for promptness and safety—for economy—for alkalinization. Made by the world's largest manufacturers of effervescent salts.

Sample and literature are at your disposal; just write

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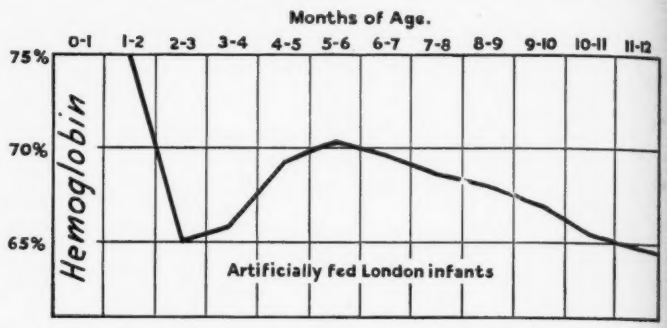
Nutritional Anemia in Infants

The accompanying chart of the hemoglobin level in the blood of infants is based on more than 1,000 clinical cases studied by Mackay. The sharp drop in hemoglobin during the early months of life has also been reported by a number of other authorities. It is noteworthy that this fall in hemoglobin has been found to parallel closely that of diminishing iron reserve in the infant's liver. The usual milk formula of infants in early life further contributes to this anemia because milk is notably low in iron. It is now possible, however, to increase significantly the iron intake of bottle-fed infants from birth by feeding Dextri-Maltose With Vitamin B in the milk formula. After the third month Pablum as the first solid food offers substantial amounts of iron for both breast- and bottle-fed babies.



Reasons for Early Pablum Feedings

1. The iron stored in the infant's liver at birth is rapidly depleted during the first months of life. (Mackay,[1] Elvehjem.[2])

2. During this period the infant's diet contains very little iron—1.44 mg. per day from the average bottle formulae of 20 ounces, or possibly 1.7 mg. per day from 28 ounces of breast milk. (Holt.[3])

For these reasons, and also because of the low hemoglobin values so frequent among pregnant and nursing mothers (Coons,[4] Galloway[5]), the pediatric trend is constantly toward the addition of iron-containing foods at an earlier age, as early as the third or fourth month. (Blatt,[6] Glazier,[7] Lynch[8].)

The Choice of the Iron-Containing Food

1. Many foods reputed to be high in iron actually add very few milligrams to the diet because much of the iron is lost in cooking or because the amount fed is necessarily small or because the food has a high percentage of water. Strained spinach, for instance, contains only 1 to 1.4 mg. of iron per 100 gm. (Bridges.[9])

2. To be effective, food iron should be in soluble form. Some foods fairly high in total iron are low in soluble iron. (Summerfeldt.[10])

3. **Pablum is high both in total iron (30 mg. per 100 gm.) and soluble iron (7.8 mg. per 100 gm.) and can be fed in significant amounts without digestive upsets as early as the third month, before the initial store of iron in the liver is depleted. Pablum also forms an iron-valuable addition to the diet of pregnant and nursing mothers.**

Pablum (Mead's Cereal thoroughly cooked and dried) consists of wheatmeal, oatmeal, cornmeal, wheat embryo, brewers' yeast, alfalfa leaf, beef bone, iron salt and sodium chloride.

[1-10] Bibliography on request.

MEAD JOHNSON & COMPANY, Evansville, Indiana, U. S. A.

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DIARRHEA

"the commonest ailment of infants in the summer months"

(HOLT AND McINTOSH: HOLT'S DISEASES OF INFANCY AND CHILDHOOD, 1933)

One of the outstanding features of DEXTRI-MALTOSE is its low fermentability and consequent preference in the management of infantile diarrhea.

In summer diarrhea, "The best food to use is boiled skimmed milk, acid skimmed milk, or dried protein milk. Carbohydrates are added in the form of dextri-maltose."—*G. Wiswell: Infant mortality and its prevention, Nova Scotia M. Bull., 15:504-509, Oct. 1936.*

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Concerning the treatment of diarrhea, "If the weight remains stationary, it is an indication that loss of substance is occurring through the stools, mostly in the form of alkaline salts. To equalize this loss of substance, the diet must be increased, but in such a way as to avoid causing fermentation. This may be done by adding dextri-maltose and preparations of protein to the food, increasing the calories until the infant is taking 100 calories per kilo. of body weight."—*H. L. Ratnoff, Nutritional disturbances, Arch. Pediat., 41:771, Nov. 1924.*

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"A very frequent cause of underfeeding results from the improper treatment of diarrhea . . . One of the greatest advances made in the science of infant feeding was the development of protein milk by Professor Finkelstein and the use of buttermilk or lactic acid mixtures. The great advantage of being able to feed the infant with fermentative diarrhea a food containing 12 calories to the ounce, like protein milk, after only one day on a starvation diet, is apparent. In addition, the further advantage of being able to safely add a carbohydrate like Dextri-Maltose No. 1 or No. 2 to the protein milk within a few days, enables one to gradually bring the infant up to its basal needs in a short time. When protein milk was first used, carbohydrate additions were advised against with the result that many children on it went into a state of collapse. The suggestion of Dr. Alan Brown of Toronto, Canada, that Dextri-Maltose be added to protein milk, was of great value."—*G. J. Feldstein: Underfeeding of infants and children, Arch. Pediat., 50:297-306, May 1933.*

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Regarding the treatment of diarrhea, "In our experience, the most satisfactory carbohydrate for routine use is Mead's dextrimaltose No. 1."—*F. R. Taylor: "Summer Complaints," Southern Med. & Surg., pp. 555-559, Aug. 1927.*

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"Again, following the teaching of the originator of protein milk, the carbohydrate added should be the one that is most easily assimilated. Dextri-maltose is the carbohydrate of choice."—*R. A. Strong: The diarrheas of early life, Mississippi Doctor, 14:9-15, Sept. 1936.*

"If the stools are acid, green, and excoriating, a food high in protein and low in fat, and carbohydrate is indicated. Dried powdered protein milk is very ideal here—one to ten dilution. On the other hand, if the evacuations are brown, watery, and stinking with putrefactive odors, a proteolytic diarrhea, it will be of advantage to add a small amount of carbohydrate, a dextri-maltose preparation being very efficacious."—*A. G. Dow: Diarrheas in infants, Nebraska M. J., 20:22-24, Jan. 1935.*

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"After the preliminary short period of starvation, protein milk should be used. . . . When the diarrhea has been sufficiently checked, dextrimaltose may be added and gradually increased until from 4 to 6 tablespoons are being used."—*W. L. Denney: Acute nutritional disturbances of infancy, Univ. West. Ontario M. J., 2:192-137, April 1932.*

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In diarrhea, "Carbohydrates, in the form of dextri-maltose, well cooked cereals or rice, usually can be handled without trouble."—*B. B. Jones: A discussion of some of the commoner types of infantile diarrhea, and the principles underlying the diets used in their treatment, Virginia M. Monthly, 55:411-415, Sept. 1928.*

SERIOUSNESS OF DIARRHEA

There is a widespread opinion that, thanks to improved sanitation, infantile diarrhea is no longer of serious aspect. But Holt and McIntosh declare that diarrhea "is still a problem of the foremost importance, producing a number of deaths each year. . . ." Because dehydration is so often an insidious development even in mild cases, prompt and effective treatment is vital. Little states (Canad. Med. A. J. 13: 803, 1923), "There are cases on record where death has taken place within 24 hours of the time of onset of the first symptoms."

In the treatment of dysentery, "As a useful supplement, Bessau's thick rice water with 1 to 5 per cent dextrimaltose may be used, or soy bean milk, which also may serve as main nourishment in the rare cases of hypersensitiveness to cow's milk."—*O. Willner: Dysentery, in The Practitioner's Library of Medicine & Surgery, D. Appleton-Century Co., Inc., New York, 1935, vol. 7, p. 371.*

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In cases of diarrhea, "For the first day or so no sugar should be added to the milk. If the bowel movements improve carbohydrates may be added. This should be the one that is most easily assimilated, so dextri-maltose is the carbohydrate of choice."—*W. H. McCaslan: Summer diarrheas in infants and young children, J. M. A. Alabama, 1:278-282, Jan. 1932.*

—•—

"When there has been a tendency to looseness of the bowels, a form of sugar which ferments but slightly, such as dextrimaltose, may be employed."—*W. Sheldon: The artificial feeding of infants, Practitioner, 134: 415-428, April 1935.*

—•—

". . . I begin to add carbohydrates slowly, by replacing ¼ ounce Casec every two days with ¼ ounce of Dextri-Maltose, preferably Dextri-Maltose Number one. As a rule, this is tolerated. When one ounce of Dextri-Maltose is used, the Casec, of course, should be discontinued."—*J. W. Reed: The etiology and treatment of diarrhoeas of infancy, Virginia M. Monthly, 53:732-735, Feb. 1927.*

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In treating chronic diarrhea, "You do not need to starve such patients, but you select a diet of dried protein milk with the addition of dextrimaltose given in small three-hourly feeds, or soured milk, or an ordinary half-cream dried milk, or a whey mixture."—*R. Hutchison: Lectures on Diseases of Children, ed. 7, William Wood & Co., Baltimore, 1936, pp. 26, 65, 71-72, 76.*

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"The effect of a combination of dextrimaltose depends on the relative properties of the two components. If maltose is in excess, it tends to cause fermentation and loose stools, while dextrins are non-fermentable. In the preparations commonly used, Mead's Dextri-maltose Nos. 1 and 2, the maltose is only slightly in excess of the dextrins, and therefore they are advantageous if there is a tendency to excessive fermentation."—*W. J. Pearson and W. G. Wyllie: Recent Advances in Diseases of Children, P. Blakiston's Sons & Co., Phila., 1930, pp. 74, 116.*

Just as DEXTRI-MALTOSE is a *carbohydrate* modifier of choice, so is CASEC (calcium caseinate) an accepted *protein* modifier. Casec is of special value for (1) colic and loose green stools in breast-fed infants, (2) fermentative diarrhea in bottle-fed infants, (3) prematures, (4) marasmus, (5) celiac disease.

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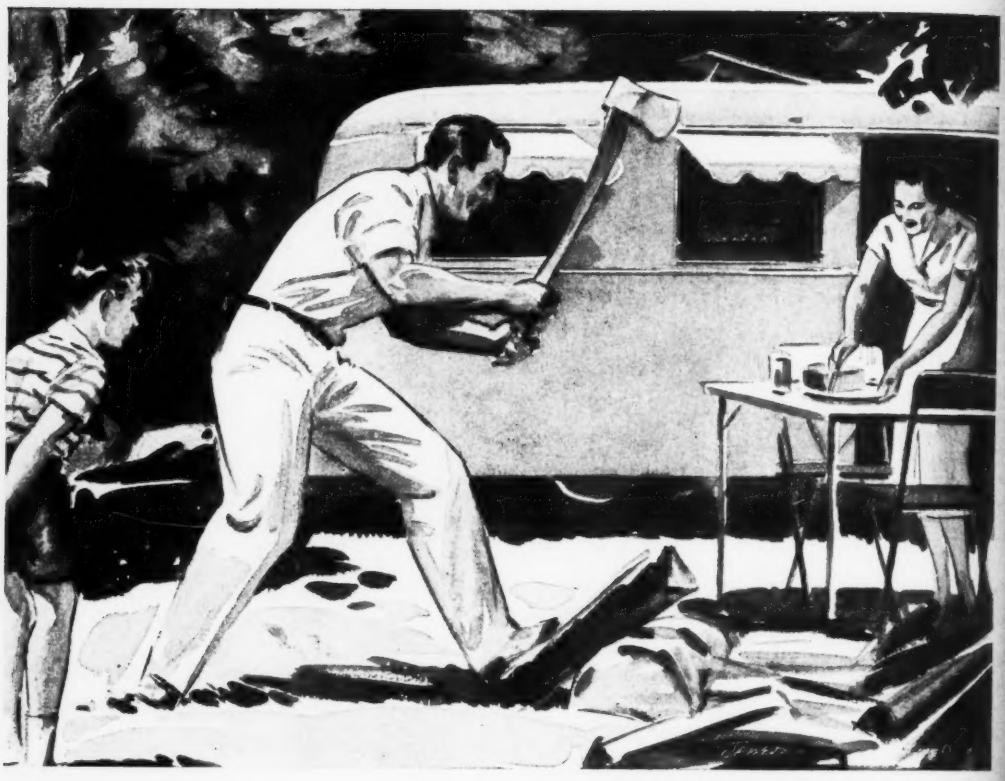

SUMMER!

Summer days show a marked increase in accidental injuries. The vacationist, the farmer, the child at play may all suffer wounds contaminated with spores of tetanus and gas gangrene-producing bacteria. Treatment of all dirt-contaminated, contused and penetrating wounds should include combined prophylaxis against tetanus and gas gangrene.

ACCIDENTS!

We suggest Parke-Davis Tetanus-Gas Gangrene Antitoxin (Combined), Refined and Concentrated. The customary prophylactic dose—1500 units tetanus antitoxin and 2000 units each perfringens and vibrion septique antitoxin—is available in syringe packages and in rubber-diaphragm-capped vials.

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